Journal

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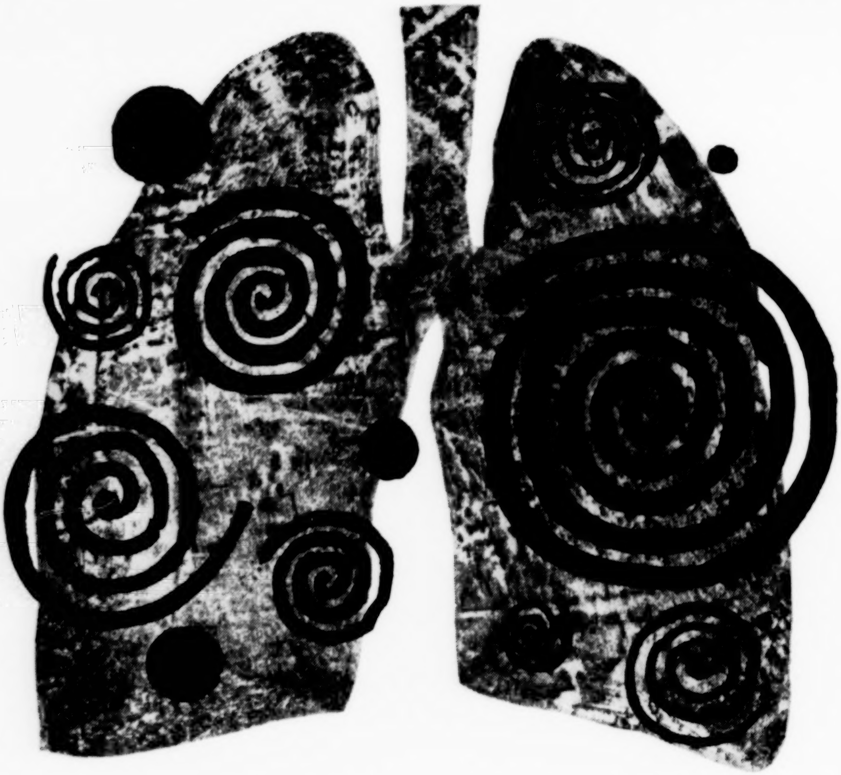

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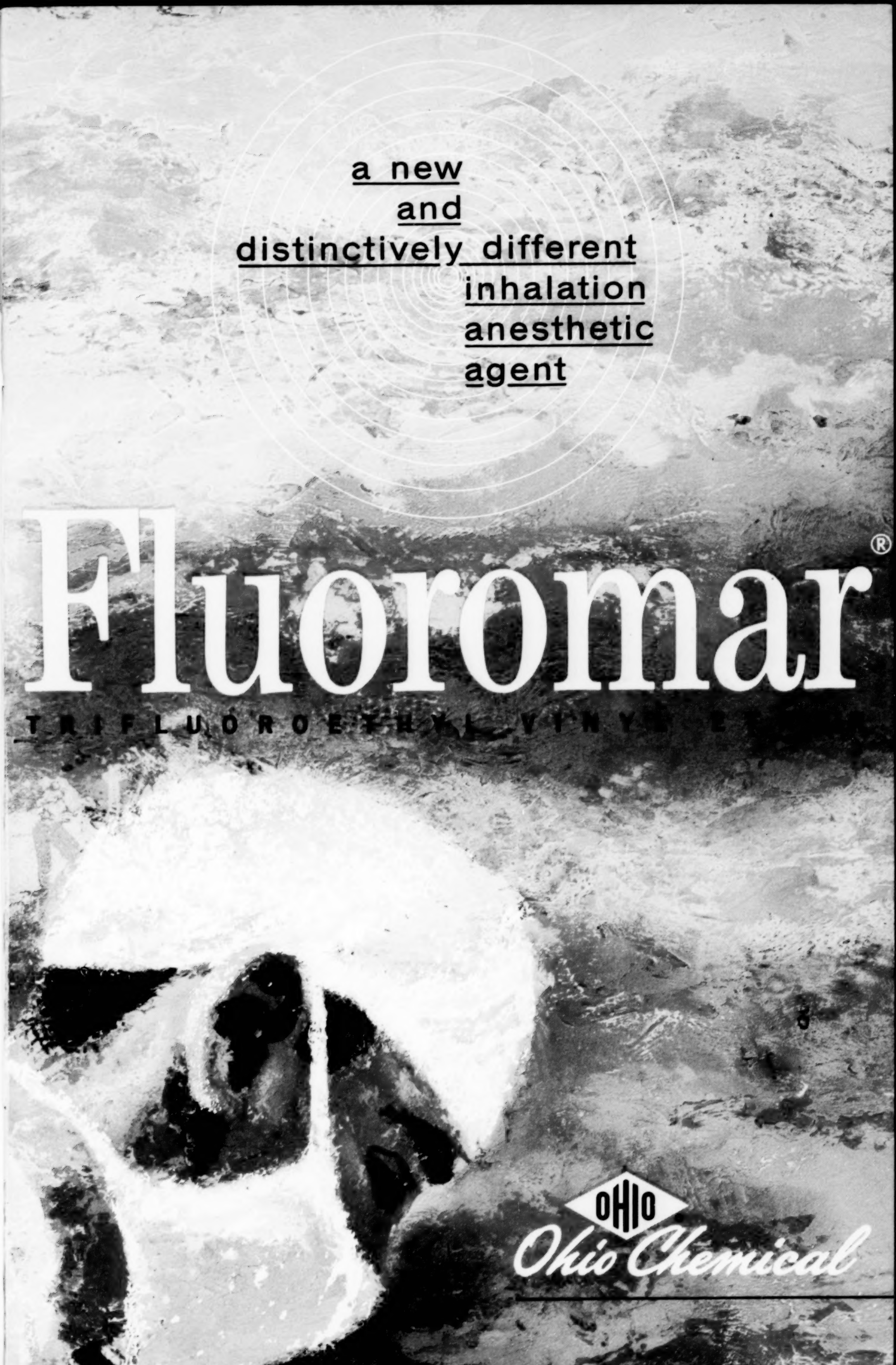

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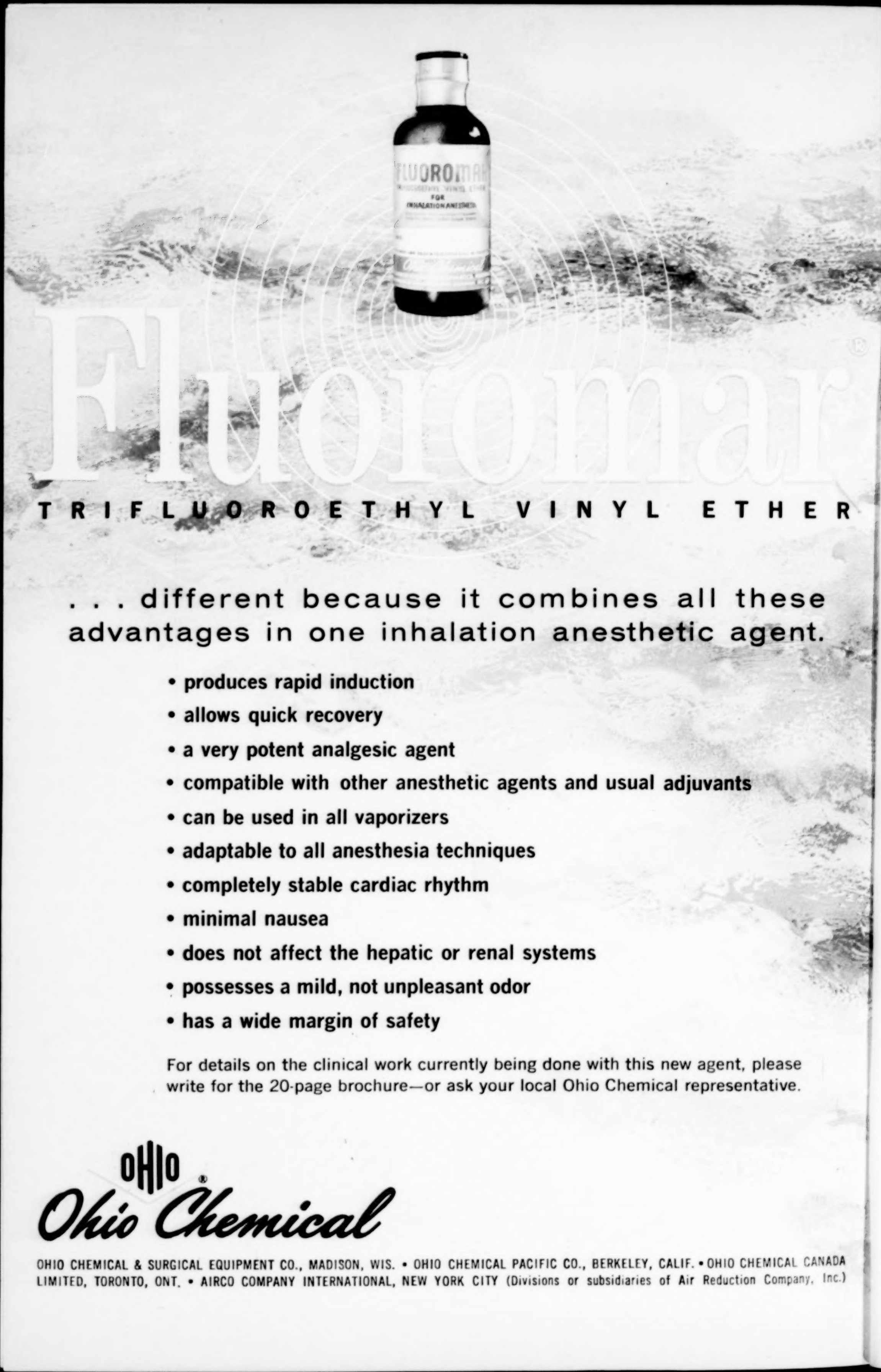

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The Journal of the American Association of Nurse Anesthetists is published bi-monthly by the American Association of Nurse Anesthetists, 130 E. Randolph St., Chicago 1, Ill. Entered as second class matter, May 14, 1945, at the post office at Chicago, Ill., under the act of March 3, 1879. Address all communications relative to editorial and advertising matters to the Editor, A.A.N.A., 130 E. Randolph St., Chicago 1, Ill. Subscription prices: to members, $2.00 a year; to non-members and institutions, $5.00 a year; single copies, $1.00. The opinions expressed in the columns of the Journal are those of the contributors and are not to be construed as reflecting the views of the American Association of Nurse Anesthetists.

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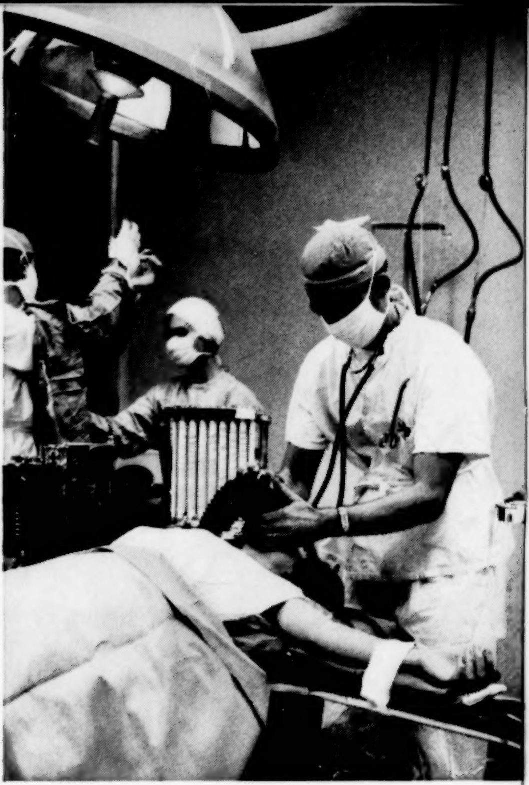

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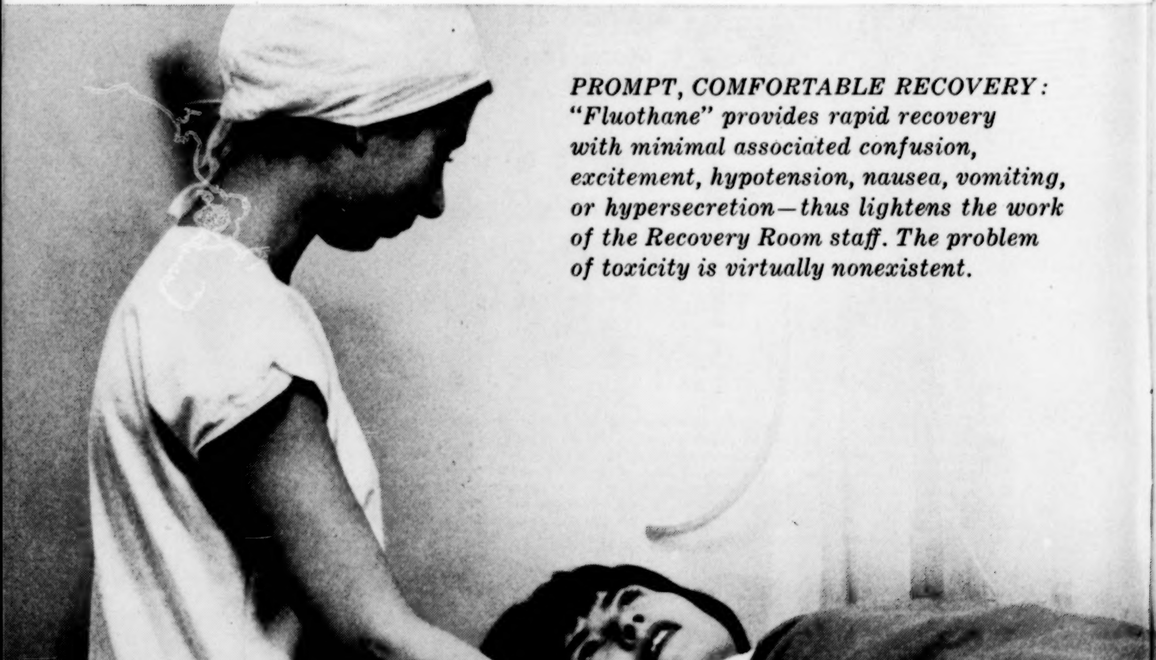

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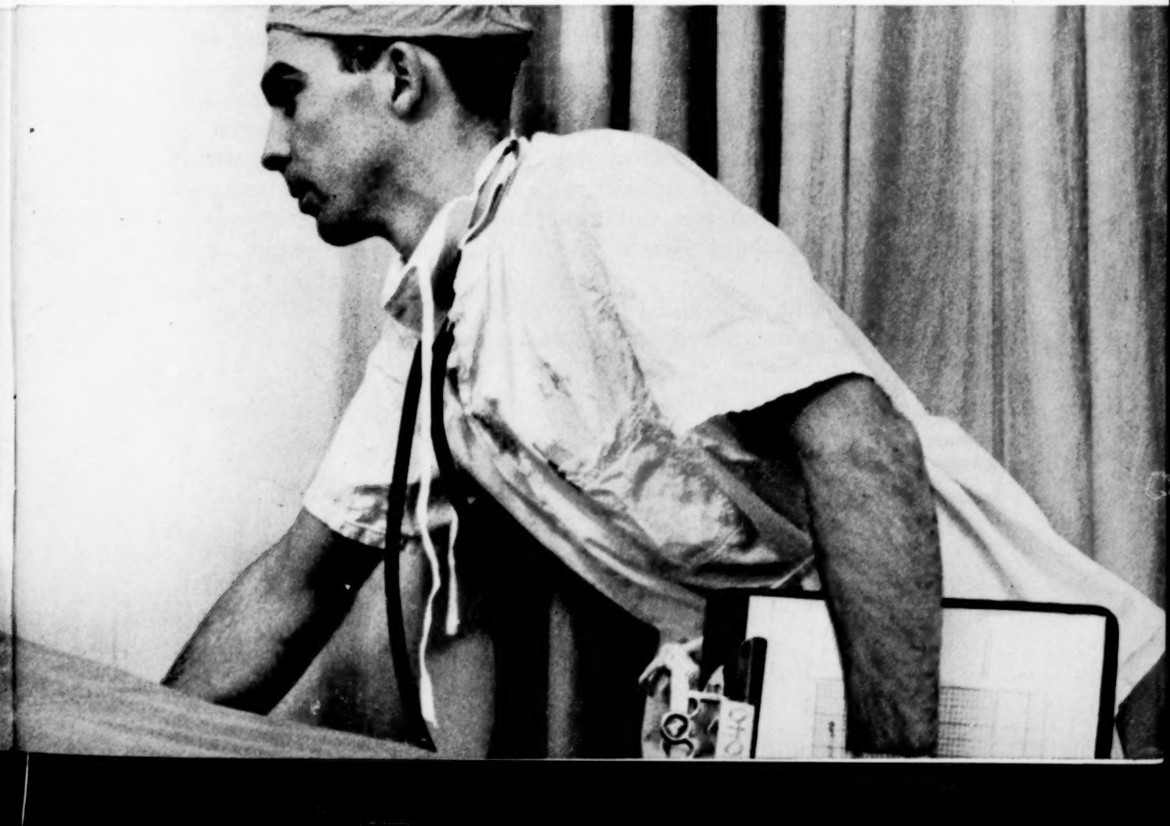

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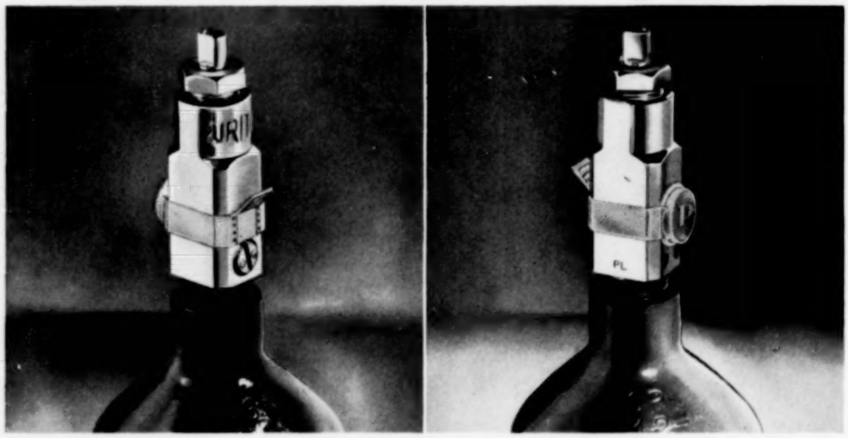

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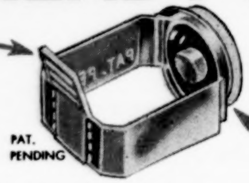
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The characteristic "NUMORPHAN effect" in premedication is well described by Appleton,[1] who used the drug in 434 patients one hour before surgery: "Patients receiving Numorphan came to operation tranquilized but without signs of confusion or disorientation.... in spite of the large number of patients who received scopolamine as part of their premedication." Nausea and vomiting were not found to be problems, an observation which confirms earlier findings[3] that NUMORPHAN is less likely to provoke the undesirable gastrointestinal reactions of morphine. Because of its sustained duration of effect, NUMORPHAN continues to provide "coverage" even when some delay in the O. R. schedule occurs.

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meets the need for supplemental analgesia

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rapid, smooth induction

NUMORPHAN facilitates rapid and smooth induction. The patient becomes capable of withstanding "the most powerful stimuli" without showing signs of being disturbed.[2] The analgesic effectiveness of NUMORPHAN is described as superior to that of meperidine in providing *supplemental* analgesia; the blood pressure usually remains within normal physiologic bounds, in contrast to the circulatory depression (often verging on shock) reported after meperidine.[2]

cough reflex retained

seldom affects circulatory and autonomic homeostasis

In a series of operations on the head and neck, patients maintained their airway and spontaneous breathing in spite of the severity of the procedure and massive bandages.[2] The patient usually remains quiet yet retains the cough reflex. The action of NUMORPHAN during anesthesia is usually characterized by the preservation of circulatory and autonomic homeostasis.[4] Where

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SUMMARY: NUMORPHAN is a highly effective analgesic providing simple sedation and tranquilization that makes possible satisfactory preparation of patients of all ages for surgery. As an adjunct to anesthesia, NUMORPHAN facilitates smooth induction and reduces the amount of anesthetic agents required.[1,2] Postoperatively, NUMORPHAN-treated patients are usually well oriented and virtually free from pain or gastrointestinal side effects; they are not drugged into deep sleep and remain in full control of vital protective reflexes.[1-4]

---

prompt response to antagonists

indicated, as with inadvertent overdosage, intravenous nalorphine (Nalline®) or levallorphan (Lorfan®) is promptly effective in reversing respiratory depression.[5]

NUMORPHAN—satisfactory in the recovery room and special care unit

continuation of operative pain-free state

fewer injections required

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NUMORPHAN analgesia accompanied by sedation, not deep sleep

NUMORPHAN is primarily an *analgesic* not a *hypnotic;* therefore the absence of deep sleep should not be interpreted as lack of effective pain relief. Regarding this unique property of NUMORPHAN, Appleton[1] has stated: "It is most important that the entire nursing service be advised of this lack of soporific effect, especially if the nursing personnel is accustomed to using narcotics in hypnotic doses..."

NUMORPHAN should not be used to induce sleep

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1. Appleton, J. C.: Anesth. & Analg. 39:505, Nov.-Dec., 1960. 2. Seigleman, M., and Wasmuth, C. E.: Cleveland Clin. Quart. 27:157, July, 1960. 3. Coblentz, A., and Bierman, H. R.: Fed. Proc. 14:327, Mar., 1955; New England J. Med. 255:694, Oct. 11, 1956. 4. Rondeau, Y.; Knaff, M., and Keeri-Szanto, M.: Union méd. Canada 90:48, Jan., 1961. 5. Adriani, J.: Postgrad. Med. 27:723, June, 1960.
*Oxymorphone hydrochloride—U. S. Pat. 2,806,033

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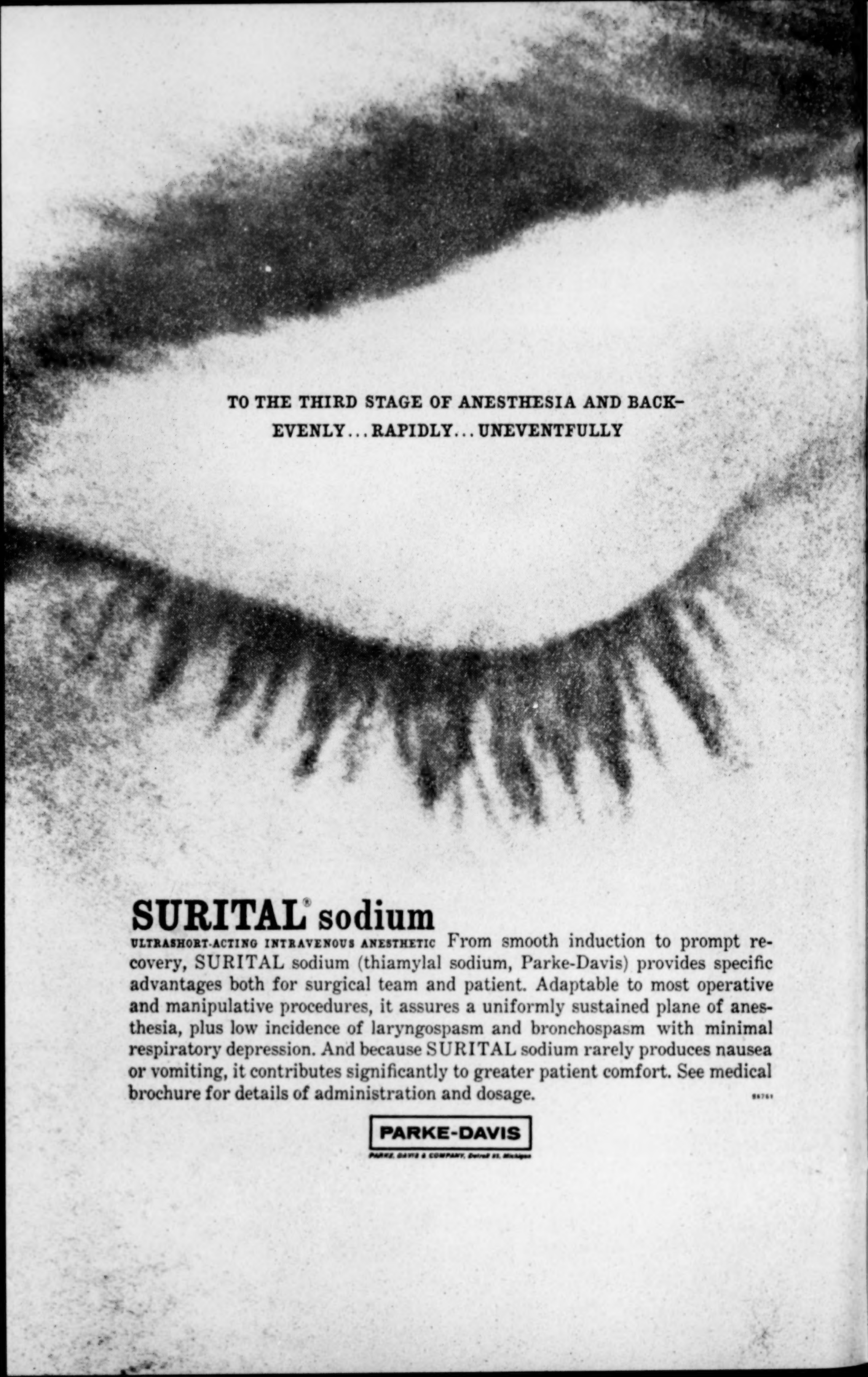

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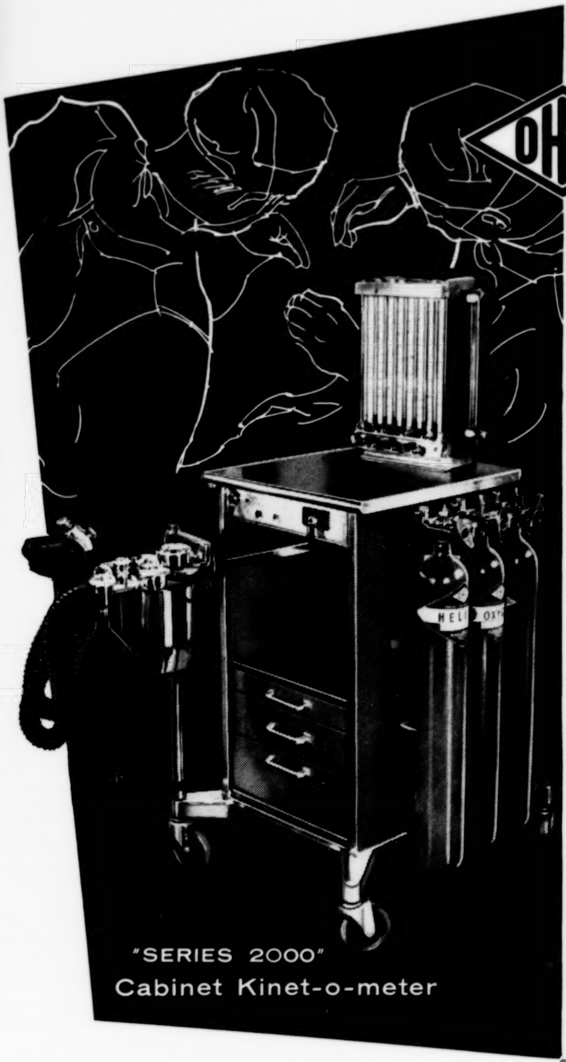

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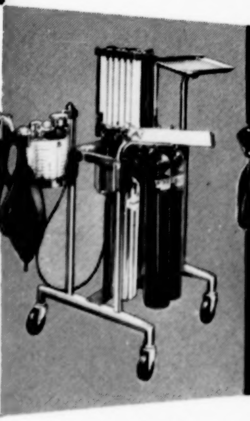

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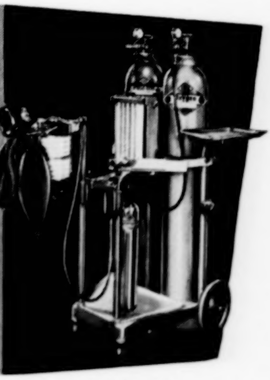

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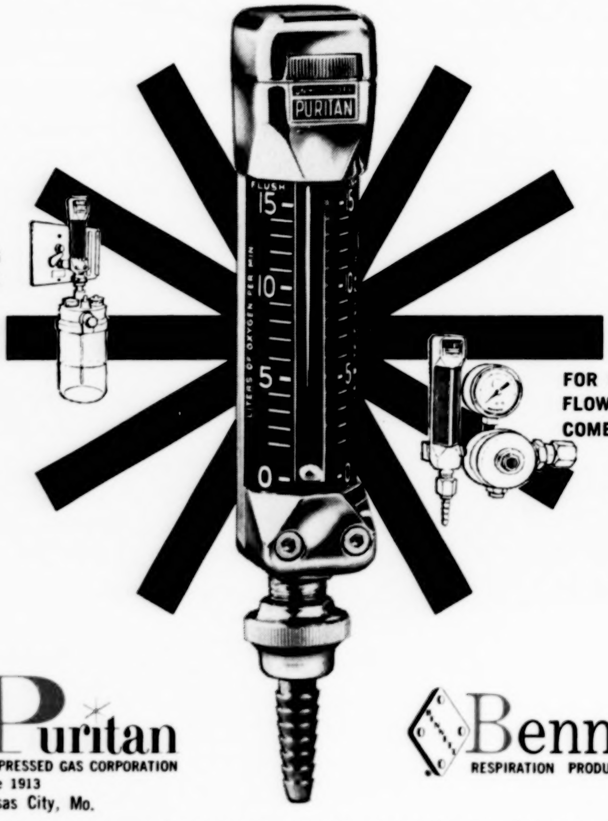

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Carbon Dioxide and Pulmonary Ventilation: Part 1

Irl T. Sell, III, Captain, MC*
San Francisco

Carbon dioxide is an odorless, colorless gas about one and one-half times as heavy as air. It is a natural by-product of many chemical processes, but occurs in nature only in minute amounts. Normally, it is found in the atmosphere in a concentration of only 0.04 per cent. Like many other substances, carbon dioxide is a natural by-product of body metabolism. Carbon dioxide is, then, a waste product, and as such must be excreted from the body. Depending upon the level of activity of the cells, that is, body metabolism and also, to a degree, the type of food ingested, carbon dioxide production parallels the consumption of oxygen by the body. If subsistence is maintained by means of a purely carbohydrate diet, the carbon dioxide output and oxygen consumption would be equal. However, since this is rarely the case and the utilization of oxygen in protein and fat metabolism is not in a direct 1 for 1 ratio, the oxygen consumption and demand of the body is usually more than the carbon dioxide production.

Various figures are quoted in scientific literature as to the amount of carbon dioxide produced and excreted each minute. Although figures below as well as above the average are given, it is generally considered that the normal adult produces about 200-250 cc. of carbon dioxide per minute. He excretes the same amount through the lungs in a concentration of about 5 per cent. The normal five pound newborn, on the other hand, produces only 12-14 cc. of carbon dioxide in comparison. Although comparative respiratory volumes differ widely, the ratio of alveolar ventilation to functional residual capacity are about the same. This demonstrates a fundamental functional similarity in spite of rate, volume, and mechanical differences.

Because carbon dioxide is a waste product and like all others produced by the body, must be excreted, a mechanism designed to eliminate it in an efficient way is necessary. Failure to excrete this product leads to abnormal increases within the cells and fluids. This may result in disturbances, frequently of a severe nature in the normal, rigidly controlled internal environment of the body. These disturbances are produced primarily by the effect of CO_2 on the acidity of the blood. The respiratory system regulates excretion of CO_2 and maintains homeostasis. Carbon dioxide then, is a gas of extreme importance in the control of respiration. It is also important in the control of circulation, producing profound effects upon acid-base balance, cerebral blood flow, cardiac conduction, body metabolism in general, and abnormal elevations may even cause a decrease in oxygenation of tissues.

---

*Anesthesiology Service, Letterman General Hospital, San Francisco, California.

Presented at the Nurse Anesthetist Workshop, Letterman General Hospital, August 26, 1960.

Carbon dioxide produced in the cells of the body by the utilization of oxygen and food is naturally found in the cells in a relatively increased concentration. Normally, intracellular carbon dioxide is found at a partial pressure or tension of 50 mm. of mercury or higher. The arterial blood coursing through that tissue normally contains 40 mm. of mercury carbon dioxide pressure. This is a difference or gradient of about 10 mm. from within the cell to the blood.

According to the law of gas diffusion across membranes, a gas which can pass through a membrane permeable to it (in this case the cell wall) will do so in the direction from a high partial pressure to low partial pressure. Carbon dioxide diffuses out of the cells into the blood. This raises the concentration in the blood and it is transported from the cells to the lungs, where the blood gives up its higher concentration to the atmosphere in the course of its passage. The blood is then recirculated through the body at its normal carbon dioxide levels. The transport of carbon dioxide in the blood is almost entirely in chemical combination with blood elements. Some, however, is carried in simple solution, but the total amount, about 0.3 ml./100 ml. of blood is small. Another small portion combines with the water of the blood and the resulting acidic products (hydrogen ion) react with the buffers of the blood to neutralize it and maintain normal acidity.

$$CO_2+H_2O \quad H_2CO_3 \quad HCO_3^-+H^+$$

Again this amount varies between 0.1 and 0.3 ml./100 ml. of blood. About 40 per cent to 45 per cent of the carbon dioxide carried by the blood is found in direct chemical combination with the hemoglobin in a form called "Carbaminohemoglobin." This bond is a loose one and in the lungs the carbon dioxide easily leaves the chemical bond and is excreted (2 ml. to 2.5 ml.). The remaining carbon dioxide produced and transported is carried in the form of carbonic acid and bicarbonate, inside the red blood cell, under the influence of carbonic anhydrase. This is an enzyme that has the ability to speed up the chemical reaction several hundred times so as to make this phase of carbon dioxide transport virtually instantaneous. This amount normally is about 2.5 ml./100 ml. of blood. As the H_2CO_3 produced from this accelerated reaction dissociates into HCO_3^- and $H+$ ions, the $H+$ ions react with hemoglobin and are buffered with release of potassium from the hemoglobin. As the HCO_3^- ions increase in the red blood cells they diffuse from the cell into plasma. The red blood cell membrane is not permeable to potassium. To maintain the same number of ions on each side of the red blood cell membrane, negative ions from the plasma diffuse into the cell. The chloride ion does this and the phenomenon is referred to as the "chloride shift" or the "Hamberger Phenomenon."

As arterial blood containing a normal carbon dioxide pressure of about 40 mm. mercury perfuses the tissue, it picks up carbon dioxide. The venous blood returning to the lungs contains a carbon dioxide pressure of about 46 mm. mercury. The alveolar carbon dioxide is lower than this (40 mm. mercury). As a result, a diffusion of carbon dioxide from blood to lungs occurs. In the process, the carbon dioxide-hemoglobin combination splits. The carbonic acid in the red blood cell, under the influence of carbonic anhydrase, again changes back to water and carbon dioxide. The carbon dioxide diffuses out. It is in this manner, then, that carbon dioxide is produced, transported, and eliminated from the body.

PULMONARY VENTILATION

Pulmonary ventilation requires the inhalation and exhalation of gas (air or anesthetic mixture). Although pulmonary exchange can be voluntary, respiratory movements are mainly controlled unconsciously and automatically by complex centers within the central nervous system. The essential area is the respiratory center which is localized to an area of specialized tissue within the medulla oblongata or hind brain. Anatomically, the respiratory center consists of two separate but closely connected areas. One causes inspiration when stimulated, the other expiration. One reciprocally inhibits the other. Both centers have connections that descend with the phrenic and intercostal nerves and with proper stimulation cause respiration. It is thought that the medullary cell masses may have an inherent rhythmicity that is influenced fairly strongly by other parts of the central nervous system above and by the vagal fibers below the center.

There are three groups of involuntary influences affecting the respiratory center:

1. The most important is the central chemoreceptors that are affected by carbon dioxide, hydrogen ion concentration, or both of the blood supplying the center. **This is the dominant mechanism for adjusting the pulmonary ventilation to the metabolic requirements of the body.**

2. Body temperature varying a few degrees above or below normal may affect pulmonary ventilation in direct response to changes in metabolism. Temperature elevations above normal may produce severe tachypnea and hyperpnea and a fall of a few degrees below normal body temperature may cause marked hypopnea, bradypnea, and apnea.

3. Ill defined and unpredictable influences may alter ventilation for varying periods of time: a. Responses to reception of pain. b. Changes associated with emotion and awareness, such as: (1) Rapid inspiration with surprise. (2) Rapid, shallow breathing associated with pleasure and, (3) Irregular sighing with anxiety.

Also affecting ventilation and thus playing a role in the control of respiration are the so-called peripheral chemoreceptors. These have been found to lie outside the central nervous system within the carotid and aortic bodies (the latter being thought not to play an important role).

Although there is still much controversy among those especially oriented in this field, recent investigations suggest that the central control of respiration is primarily by the medullary respiratory center. It is influenced by changes in the acidity of blood from the dissociation of carbon dioxide, and from the molecular carbon dioxide as well. The peripheral chemoreceptors are thought to affect respiration by reflex stimulation of the central respiratory center. These have been shown to be stimulated by decreased oxygen tension rather than by the carbon dioxide or hydrogen ion mechanism. The possibility of an H+ ion mechanism exists in relation to an anaerobic type of metabolism in these centers, producing increased H+ ions. Thus, changes in acid-base balance by virtue of the action of hydrogen ions (acid) on the blood bathing the respiratory center and changes in the oxygen tension surrounding the peripheral centers appear to be the primary factors in control of pulmonary ventilation. Anything affecting availability of oxygen, production of carbon dioxide or the respiratory center itself, produces an effect on pulmonary ventilation. It is known that the respiratory center in the medulla is unresponsive to mild hypoxia and depressed by severe hypoxia. Conversely, the chemoreceptors are somewhat sensitive to increases in carbon dioxide, but quite sensitive to decreased oxygen tension.

In short, the task of pulmonary ventilation actually is two-fold:

1. To maintain equilibrium between oxygen demand and supply.
2. To maintain acid-base balance (carbon dioxide homeostasis).

ACID-BASE BALANCE

The term "acid-base balance" is a difficult one to understand because of the magnitude of scope, and complexity of make-up. We need not be experts on the subject, however, we should have some idea of its significance. This is especially true of anesthetists because of the ease with which changes can unknowingly or purposely be created. In simple terms, acid-base balance means regulation by the body of the hydrogen ion concentration in body fluids. Hydrogen ions come from acids. When the H+ ions increase by increased acid production the tissue fluids get more acid. When the H+ ions decrease they get less acid. To combat this, certain elements within the body react with the excess H+ ions to neutralize the acid and bring the blood back to its normal reaction.

To prevent this increase and decrease in H+ ion concentration from whatever cause, three special control systems have evolved within the body:

1. Acid-base buffer system. A buffer is simply a solution of two or more chemical compounds that prevent marked changes in the acidity of the fluid in which they are found. There are several within the blood, resisting or buffering any sudden changes in acidity that might otherwise be fatal. The carbonic acid-bicarbonate buffer system is the most important.

2. Respiratory mechanism. With an increase in the H+ ion (and/or carbon dioxide) in the fluids, stimulation of the respiratory center occurs and the body responds with an increased ventilation. How does this work? The more rapidly and the more deeply a person breathes or is breathed, the lower will be the partial pressure of carbon dioxide in his alveoli. The rate at which the alveolar carbon dioxide is moved from the lungs to the atmosphere is proportional to the ventilatory rate of the lungs. Also, the lower the partial pressure of carbon dioxide in the alveoli, the smaller becomes the quantity of carbon dioxide dissolved in the blood and body fluids. As we have seen, when carbon dioxide dissolves in body fluids, a constant proportion of it combines with water to form carbonic acid. Thus a person breathing rapidly or being hyperventilated will lower the carbonic acid

in body fluids, hence the fluid becomes less acid, or alkaline. On the other hand, breath-holding, apnea, hypoventilation, or rebreathing carbon dioxide, as the case may be, increases the carbonic acid content in blood and fluids. Fortunately, most of the carbonic acid formed is buffered by the other buffer systems. Even so, excess carbonic acid can exhaust body buffer systems and produce severe acidosis if the apnea or hypoventilation is prolonged.

3. Renal mechanism. When H+ ions (or acidity) changes from normal, the kidneys excrete an acid or alkaline urine to readjust the H+ ions toward normal.

Of these three protective mechanisms, the buffer systems can act in a fraction of a second to prevent marked changes. The respiratory mechanism takes several minutes to readjust normally. Although the kidney is perhaps the most important regulatory system, it requires several hours, or even days, to adjust to marked changes in the body fluids. The buffer systems and the respiratory mechanism comprise the **moment to moment control over acid base balance.** In other words, they control pulmonary ventilation from moment to moment.

There are four types of disturbances in acid-base relationships, all but one of which affect pulmonary ventilation directly:

1. Respiratory acidosis
2. Metabolic acidosis
3. Respiratory alkalosis
4. Metabolic alkalosis

In respiratory acidosis the defect is retention of carbon dioxide and results from an inability, from whatever cause, to excrete carbon dioxide in adequate amounts by way of the lungs. Causes for this condition include diminution in depth of respiration as from primary depression of the central nervous system, from drugs, and paralysis of respiratory muscles (muscle relaxants); reduction in functioning pulmonary parenchyma, such as pneumonia, atelectasis (tube in bronchus), hydrothorax or pneumothorax; or interference with the exchange of gases across the alveolar membranes as in pulmonary edema, pulmonary emphysema; rebreathing with poor carbon dioxide absorption (spent absorbent, poor packing), faulty valves, increased dead space. This distortion in acid-base equilibrium can be improved by correction of the primary defect.

As respiratory acidosis progresses, the carbon dioxide content of the fluids and thus the acidity increases. The respiratory center responds, being remarkably sensitive to changes in acidity causing increased ventilation (if not depressed by drugs). This tends to lower carbon dioxide by excretion through the lungs. However, if the disorder is progressive, that is, if carbon dioxide excretion is less than production, the respiratory center becomes less sensitive to the high tensions of carbon dioxide. This is by direct depression of the center and it may actually interfere with ventilatory exchange. It has been suggested that techniques of artificial respiration such as the use of a mechanical respirator to increase ventilatory exchange be used to augment the excretion of carbon dioxide. This would decrease the tension of the gas in extracellular fluid, and allow the respiratory center to regain its normal sensitivity to lower tension of carbon dioxide.

Metabolic acidosis results from accumulation of (1) fixed acids due to excess production of acids (diabetes). (2) an inability to excrete acid properly by the kidneys, or (3) the administration of large acid loads. This condition, also by increasing carbon dioxide tension, increases respiration by respiratory center stimulation and tends to reverse the situation. It is said that severe respiratory acidosis may lead to some degree of metabolic acidosis.

Respiratory alkalosis is produced by excess elimination of carbon dioxide through the lungs by over-ventilation, whether consciously or by hyperventilation under anesthesia by the anesthetist. In the awake individual this excess blowing off of carbon dioxide frequently produces symptoms usually described as dizziness or light headedness. Other complaints include circumoral paresthesias, numbness and tingling in fingers and toes, sweating, palpitation, tinnitus and tremulousness. It may lead to frank carpopedal spasm. The mechanics for this is not clear, although there is thought to be a decrease in ionized calcium but determinations have not always borne this out. However, under anesthesia none of these are present and indeed there are no signs of this condition other than apnea, as a rule. There is also no evidence that hyperventilation per se is harmful to an individual. Indeed, modern techniques of anesthesia utilizing hyperventilation as a means of reducing brain mass for intracranial surgery is quite effective and apparently has no deleterious effects on the patient. On the other hand, it greatly facilitates exposure for the surgeon.

The mechanism is, of course, the decrease in cerebral fluid by decreased cerebral blood flow. Metabolic alkalosis may arise in two ways: by the ingestion of large amounts of bicarbonate or loss of H+ ions in excess from the extracellular fluid (as in protracted vomiting). The body can compensate for this to a degree by diminished respiratory exchange. It is the former type of derangement of acid-base balance, or prevention of it, in which anesthetists are primarily interested, for it is through the respiratory system that anesthesia-producing drugs are administered. In doing so, and with ancillary drugs affecting respiration too, we must be overly cautious and diligent in our attempts to maintain the adequacy of respiration and thus carbon dioxide homeostasis.

S. M. Tenney, discussing the interpretation of respiratory drug effects in man, states that for normal man, breathing air at sea level, respiratory homeostasis is best defined in terms of alveolar ventilation. This is the amount of air in contact with alveoli. If one knows the amount of carbon dioxide exhaled per minute and the concentration exhaled, the alveolar ventilation can be calculated. If these are not known, the alveolar ventilation can be deduced by subtracting the dead space from the tidal volume. A ready and reasonable estimate of dead space is derived by considering it to be 1 ml./pound of body weight. It is this value that gives the anesthetist the most accurate indication of the effectiveness of pulmonary ventilation.

For any metabolic carbon dioxide output, partial pressure of carbon dioxide of alveolar air, or arterial blood, varies inversely as the alveolar ventilation and represents the most ready index of change in effective ventilation. Since knowledge of effective alveolar ventilation and change is the best indication of respiratory stimulation or depression, many results reporting change in minute volume or rate are misleading. For example, the rate may be increased at the expense of expired volume (tidal) and what appears to be a stimulatory effect may not be so when the dead space is the only thing being ventilated more. One must exercise considerable caution in the interpretation of studies using these indices of respiratory response.

Padget, as early as 1928, studied the respiratory response to carbon dioxide in normal males and concluded:

1. When air containing an increased amount of carbon dioxide is breathed, the maximum increase in respiration occurs only after the mixture has been breathed for a time. The length of the lag time varying with the concentration of inspired carbon dioxide.

2. The reaction of any one subject to the same concentration of carbon dioxide at different times is, in general, constant. The reaction of different individuals varies greatly.

3. When inspiring carbon dioxide mixtures, there is no delay in raising the carbon dioxide tension of the arterial blood. From this, it appears that the lag in respiratory response may be explained by the time required to saturate tissue, especially the respiratory center, with new carbon dioxide tension. Or it may be that the initial increased carbon dioxide is buffered with only minute initial pH changes. Normal unanesthetized subjects can increase their minute respiratory volume by increasing tidal volume as well as rate, in such a way as to parallel the carbon dioxide being inspired so that blood concentrations of carbon dioxide is not increased appreciably. This is not always the case with the anesthetized subject.

The effect of carbon dioxide on normal human subjects has been noted throughout the years by various people. A summary of the effects follows:

1. Usually, 0-2 per cent carbon dioxide produces no awareness of increase but may be reflected in increased respiratory minute volume (increased tidal).

2. Concentrations of 2-4 per cent carbon dioxide cause awareness of increase but is tolerable. Breathing may become labored but dyspnea does not ensue. Mental depression, chilliness, and persistent headache may occur. The pulse rises and minute volume increases.

3. The limit of tolerance for long exposure is 4-6 per cent. There may be further mental depression, headache, dizziness, nausea. Minute volume further increased.

4. Six to 8 per cent is tolerable but very unpleasant, even for moderate exposure. The respiratory rate increases up to 30 per cent. The respiratory minute volume may increase 225 per cent. The pulse may increase 15 per cent or more. Systolic blood pressure may increase 25 per cent, and the diastolic may do the same.

5. Eight to 10 per cent is near the limit of tolerance because of intense dyspnea. Ten per cent is considered the absolute upper limit. It is likewise here that one may see the maximum changes in respiratory response and circulatory response to carbon dioxide. The respiratory rate may be 40 per cent higher than normal. The respiratory minute volume may be three times normal (275 per cent). The pulse may be 30 per cent higher. The systolic blood pressure may be

35 per cent higher, as well as the diastolic.

6. Concentrations between 10 and 12 per cent produce unconsciousness if prolonged over 10 minutes. This is intolerable after 2 minutes. The first breath is unpleasant, with decreased vision, flushing, sweating, dizziness, and fatigue; prolonged headache. Here we reach a critical level since further increases result in no further stimulation of respiration, pulse, and diastolic pressure although the systolic pressure continues to increase with increasing concentration of carbon dioxide. Above this level, carbon dioxide becomes narcotic and depressant in its effect on the individual.

7. Concentrations between 12 and 15 per cent are tolerable only for a minute or so.

8. Fifteen to 25 per cent carbon dioxide is irrespirable, except for a few seconds. It may produce laryngospasm, whooping inspiration, throbbing in the head, mental dullness and intolerable dyspnea and cyanosis.

9. Concentrations above 25 per cent produce complete narcosis and may produce convulsions either during or after exposure.

According to Seevers, those who have never attempted to respire 5 per cent carbon dioxide for an extended time have missed a disagreeable experience. If one is able to breathe, voluntarily, 10 per cent carbon dioxide for more than a few moments, he possesses more fortitude than most. Yet, the scientific publications are full of good evidence that we expose patients under anesthesia to these and even higher concentrations of carbon dioxide with some regularity. Widespread use of muscle relaxants, depressant drugs in connection with anesthetic drugs, themselves depressant, and relatively modern closed techniques of administration have no doubt increased the incidence of such exposure and not always to the advantage of the patient. Since patients under anesthesia differ from normal human subjects in that they are no longer capable of objecting to the disagreeable procedure of respiring high concentrations of carbon dioxide, their biochemical and physiologic resources must be called upon to compensate for this threat to their security. If these resources have been reduced during recent bouts with disease the added insult may be too great. If the respiratory mechanism cannot respond because of depression from some cause, or if the patient is forced to re-breathe a portion of his last breath, an increase in blood carbon dioxide with its effects on acid-base equilibrium is inevitable.

Part II will be published in the August issue of the Journal.

Some Practical Aspects of Pediatric Anesthesia

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In pediatric anesthesia there are several adages which we feel are in error. One is that open drop anesthesia is the best and only way to manage infants and children. Another is that the less premedication the better and that here true homeopathy is the safest and the best. Another is that children are just little adults and can be managed in the same way. We feel that these old chestnuts should be revised in light of what we feel are better ways of handling pediatric patients.

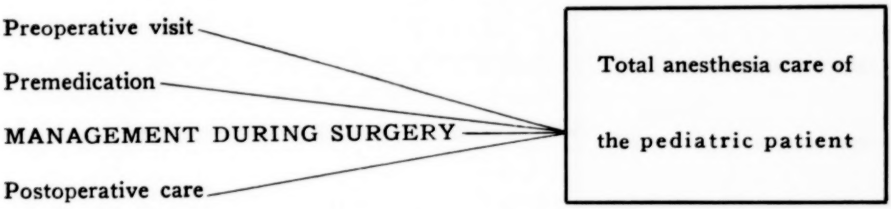
I will discuss some methods of management and the reasons for using these methods. Some aspects of the full range of treatment of the pediatric patient will be presented beginning with the preoperative visit and ending with transfer of the child to the recovery room.

PREOPERATIVE PERIOD

Table 1 indicates that the operative period is just one phase of anesthesia management of the child. Admittedly it is the most important phase from our viewpoint as anesthetists, however, we must consider anesthesia as starting at the time of the preoperative visit.

The preoperative visit to the child is a must. At the time of this visit it is important to explain to the child what is to be done, how it is to be done and the fact that he will be treated gently. It is important to avoid being too literal in explanation. Use references to the child's knowledge of stories and events to illustrate what will happen. He is told that the preoperative shot will be a small one and that the medicine given before the shot may in fact keep it from being felt at all. The child may be told that he will blow up a magic balloon to induce a state of sleep, that he may feel silly and

Table 1.


Preoperative visit
Premedication
MANAGEMENT DURING SURGERY
Postoperative care
Total anesthesia care of the pediatric patient

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Presented at the annual meeting, Mid-South Assembly of Nurse Anesthetists, Memphis, Tennessee, February 17, 1961.

giggly while he smells something which will not smell bad, will not smell "stinky" but will smell sort of sweet. He is told that his sleep is like the Sleeping Beauty of fairy tale fame and that he will wake up when we are ready but that until then nothing can hurt him or awaken him. These and other such methods of approach will soften the memory of the anesthesia and of the operative day and will help in conjunction with proper premedication, to eliminate the operative day from the memory of the child.

Table 2.

Premedication requirements vary depending upon many factors

1. **Weight — not age**
2. **Emotional state—the stoic requires less than the very active child**
3. **Physiological state**
   - **Respiratory function**
   - **Circulatory efficiency**
   - **Endocrine function**
4. **Pathological state**

The dose of drug used for premedication depends on several factors. These are shown in Table 2.

We cannot set up a chart and expect the patient to conform to it. We must tailor the dose to the particular child. In the healthy child of usual activity one can refer to the table 3[1]. As is seen, the drug dosage depends not on age but weight and is modified by physiological status. We consider premedication adequate when we can easily awaken the child from a state of peaceful sleep into which he drifts if undisturbed. We use scopolamine in all pediatric ages, alone for the infant, combined with a medium or short acting barbiturate for those from about 9 months to 2 years, and with a barbiturate and Demerol for older children. This is modified depending on the vitality of the child. On a practical everyday level we find the following points on premedication to be among the most useful:

1. Avoid "on call" medication. Time the administration of drugs so that barbiturates are given one and one-half hours preoperatively, belladonna drugs are given one hour preoperatively and opiates 1 hour preoperatively.
2. If the child cannot swallow capsules, i.e., nembutal, do not insert them rectally. Too often the capsules are not melted for a long time and the drug is not absorbed until long after it is needed. This may result in a puzzlingly deep patient during surgery. To avoid this problem, dissolve the powder in 1 oz. of

Table 3.
Percentage of Adult Dosage Applicable to Patients of Varying Weight

Weight kg.	**Weight** lb.	**Surface Area in Square Meters**	**Percentage of Adult Doses**
3	6.6	0.2	12
6	13.2	0.3	18
10	22	0.45	28
20	44	0.8	47
30	66	1	60
40	88	1.3	76
50	110	1.5	88
65	143	1.7	100
70	154	1.76	103

> water and using an Asepto syringe and catheter, inject the solution rectally, taping the buttocks together after administration.

For this purpose we use a tray consisting of small emesis basin, Asepto syringe and two whistle tip catheters, one size 8 or 10, the other size 12 French. We use the small catheter to wash out the stomachs of Caesarean section babies and the larger catheter to inject drugs rectally when the occasion arises.

We never take a child into the operating room struggling, crying and generally upset unless emergency conditions make it impossible to avoid this. We give supplementary sodium pentothal rectally in doses of from 8 to 10 mgm. per pound of body weight in 1 ounce of water, and wait until the child falls asleep. This ordinarily takes from 5 to 10 minutes. We then induce anesthesia after the child is asleep. We feel that expediting the surgical schedule, per se, is never more important than the undesirable effects resulting from forcibly holding and restraining a struggling child. The fears thus developed may remain unresolved for years. Ten minutes wait is not unreasonable occasionally, if it prevents such fears in the mind of the child. We feel that the importance of psychological preparation and premedication in pediatric anesthesia cannot be overemphasized.[2]

Recently we hear much of monitoring the various physiologic parameters. The simplest and one of the most effective monitors is the stethoscope taped to the precordium. Simultaneously one can hear heart and breath sounds and thus check both.

INDUCTION

In the induction of children we use cyclopropane exclusively. We hold the mask five or six inches from the face distracting the child with talk of dogs, cats, popsicles, lollipops and whatever holds the interest of the child while induction is progressing. Only rarely do we have more than a momentary sign of objection to induction. Gradually we bring the mask closer to the face using a high cyclopropane flow rate and then after the mask is upon the face induce anesthesia rapidly with a 50% cyclopropane and 50% oxygen mixture. At this time respirations are vigorously assisted and intubation can be accomplished after one to four minutes with little or no bucking or coughing. At times we use Surfacaine jelly as a lubricant-anesthetic. We have had no postoperative difficulty following the use of Surfacaine. After induction we employ endotracheal anesthesia for all otolaryngological procedures except the removal of pulmonary foreign bodies or glottic tumors requiring endoscopy.

Tables 4, 5 and 6 present the advantages and disadvantages of endotracheal versus pharyngeal insufflation anesthesia.[3]

For diagnostic bronchoscopy we intubate the trachea with a long, small tube beside which the bronchoscope is passed. Because of air leak with the bronchoscope in place during endoscopy, high flow rates are necessary for adequate exchange. We have found, for this purpose and for general use, that Portex tubes provide flexibility, thin wall, resilience, mould themselves to contour without trauma and are easily cleaned. One word of caution, Portex and some other plastic tubes become very hard when soaked in some bacteriocides and should be discarded before they become hard. We use Portex tubes for almost all of our endotracheal anesthesia for pediatric patients ranging from tracheo-esophageal fistula in prematures to the largest adolescent. In choosing the proper size, pick one of the expected size, one larger and one smaller. If the tube will go

Table 4.

Disadvantages of Pharyngeal Insufflation Anesthesia

1. Unless only minimal preoperative sedation is given, induction is prolonged.
2. Laryngospasm, breath-holding and hypoxia frequently accompany the unpleasant, prolonged induction period.
3. Often times, with the blanketing of the open drop mask with towels to hasten induction, there is reduced oxygen and increased carbon dioxide in the inspired air.
4. As the depth of the anesthesia increases, there is progressive hypoventilation.
5. Choice of agents is limited to those which vaporize on the open drop mask.
6. Everyone in the operative team breathes ether vapor.
7. Airway obstruction is unavoidable unless special positions, gags and attention to airway are provided.
8. There is no control of the airway or of the pulmonary ventilation.
9. Deep levels of anesthesia are required and a stable level of anesthesia is difficult to maintain.

Table 5.

Advantages of Endotracheal Anesthesia

1. More sedation can be administered preoperatively without fear of uncontrolled respiratory depression. The patient thus is more cooperative and comfortable.
2. Lighter stages of anesthesia may be employed during the operation.
3. Any type mouth gag can be used without fear of respiratory obstruction.
4. Dead space is reduced.
5. A patent airway can be maintained in spite of accumulation of blood or secretions in the pharynx and in spite of positional changes.
6. Pulmonary ventilation can be assisted or controlled.
7. Secretions can be removed easily.
8. The anesthetist can work safely away from the operative field.

Table 6.

Disadvantages of Endotracheal Anesthesia

1. The tube may become obstructed by tenacious mucus or blood, by kinking or by clamping of the teeth upon the lumen of the tube. Obstruction may result from using a tube which is too long.
2. Mechanical problems may arise—that is inadvertent separation of endotracheal tube from connector, the metal-to-metal fittings may become ill-fitting and leaking. The tube may be cut by the surgeon.
3. Laryngoscopy may result in chipped or dislodged teeth. Lip or tongue may be cut.
4. The tube may be too short or too long. This is usually the result of trying to "make do" with equipment that is not properly selected.
5. Glottic or subglottic edema may result from the use of lubricants, too large bore tubes or dirty tubes.
6. Maintenance with too high a positive pressure may result in rupture of alveoli or diminished pulmonary blood flow.
7. Ill chosen tubes or fittings may result in partial obstruction through diminished lumen.
8. Granuloma of the cords may occur in adults, but is seldom if ever seen in children.

through the nostrils it will almost invariably slide readily between the cords.

MAINTENANCE

Two very important factors in the smooth management of children are vigorous gas exchange and the other resistance free exchange. We assist or control all but a few children. Some patients will not permit assistance or control without resisting, however this is quickly determined by trial. We intubate and control all tonsillectomies to prevent aspiration as well as to insure adequate exchange. Assistance or control should be full, rapid and smooth. It should not be jerky. There is a fine point at which exchange is too vigorous and produces traumatic overinflation, and another at which exchange is too small. These differences are learned by careful attention to management and by constant self-criticism.

Some surgeons complain of the tube getting in the way and won't try to work with endotracheal anesthesia because of this psychological block. There is adequate room with the tube in place and with practice it is seen that the tube does not get in the way at all.

We prefer Foregger to and fro equipment because it is easily cleaned, sizes are available for all age groups and because there are many adapters available for use with the equipment. It is simple, almost foolproof and is very readily cleaned. It is very important to use flow rates with permit opening the "rat tail" on the bag or to permit frequent dumping of the bag. This makes sure carbon dioxide removal is adequate.

We discard soda lime after any case lasting more than 45 minutes. This is admittedly wasteful however we want to make positive the absorption is efficient. It is important to blow all of the dust out of the cannisters. A little puff through the cannister with mask in place will not remove the soda lime dust. The proper method is to apply the mouth to the cannister and blow hard through it several times to blow out the dust.

The different techniques of management are too numerous to describe at this time, however we prefer to use the circle filter in those children of the size of the usual healthy eight year old and to use non-rebreathing techniques or to and fro techniques below this size.

In all pediatric age groups we assist the respirations vigorously and at times control respirations for some procedures. We believe that the muscle relaxants should be used when indicated by the operative conditions rather than by a set of predetermined inflexible rules. One general rule for endotracheal anesthesia is that if a clear airway is not very easy to maintain, intubate immediately. We are constantly bombarded by Madison Avenue and sales slogans these days such as "Motorists wise Simonize". If there be one for us it might be "Anesthetists who cerebrate, readily intubate". Too often the anesthetist waits until airway difficulty has progressed from minor obstruction to serious hypoxia before intubating in a final attempt to produce adequate exchange. This comes through fear of intubation as a procedure or because of real or imagined complications of intubation. These complications need not exist. Technical skill can be acquired through instruction and experience. This problem of semi-asphyxiation need not occur if intubation and proper maintenance are used instead of struggling to maintain a clear airway, by means of a poor fitting mask or in the presence of an unusual facial contour.

It is important to carry the pediatric patient just deep enough for the operative procedure and no deeper. We carry our pediatric patients very light, just short of bucking, coughing or straining. For this reason it is doubly important to make sure the premedication with a belladonna type drug is given well in advance of surgery. These patients carried in lighter planes of anesthesia quickly reflect the adverse effects of stimulus when those in deeper stages would not show these effects. The manifestations of stimulus are usually shown as bradycardia, hypotension, (at times hypertension may ensue instead) and sweating. At times those patients carried in lighter planes of anesthesia will retch and vomit at the time of stimulus, having been quiescent previously. The belladonna drugs will block bradycardia and hypotension resulting from a stimulus. They will not block retching and vomiting.

A very troublesome feature of light states of anesthesia at the time of extubation is laryngospasm. We have avoided this by not suctioning the tube during withdrawal as we ordinarily do following adult management. We carefully clean the mouth and tube lumen preliminary to extubation, assist respirations vigorously during a few breaths of oxygen inhalation and then apply pressure to the bag while withdrawing the tube. This forces secretions up into the mouth with the tube and doesn't let them remain in the trachea following extubation. With the use of this technique we have had no further difficulty with extubation laryngospasm and have not seen postoperative atelectasis.

Should glottic edema occur following extubation, cold steam via Mistogen or Croupaire units will quickly clear this difficulty. Our problems with postoperative glottic edema have arisen in children requiring bronchoscopy for removal of foreign bodies. Cold steam is ordered routinely postoperatively in these children and so far in many children we have not had to do tracheotomy.

Our most important troublesome postoperative problem is tracheostomy care on those with neurosurgical problems or caustic burns requiring tracheostomy. Unless a few cubic centimeters of normal saline are dropped into the tracheostomy opening several times daily, crusts form in the trachea and in the tube. With variations in nursing ability of our present day shifts of nurses we have had near catastrophe on several occasions. We take each nurse who is new to us through the routine of tracheostomy care as she spends time in the Intensive Nursing Care Unit (INCU) and in this way have avoided further trouble with tracheostomy tubes.

Square cut or whistle tip catheters are the only types which should be used in suctioning endotracheal tubes or tracheostomy tubes. We prefer whistle tips because square cut catheters sometimes cut the mucosa of the trachea. We find that the straight French catheter simply doesn't do the job and we believe that they should be forbidden in recovery rooms or in anesthesia equipment drawers for purposes of suction. Other features of postoperative care such as positioning, respiratory depression and routine fluid needs should be familiar to anyone here.

SUMMARY

The total care of the pediatric patient begins with the preoperative

(Continued on page 172)

Reflex Hypotension

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Hypotension during the administration of an anesthetic is a frequent and, at times, a perplexing complication. As one anesthesiologist has stated, "The patient in shock is almost never a proper candidate for surgery or anesthesia."[13] Although this reference was primarily to the preoperative patient, it is also applicable to the patient during the course of an anesthetic. Severe hypotension occurring during surgery may, at times, necessitate a delay in the surgical procedure until the hypotension can be remedied.

In speaking of hypotension, there are numerous factors which can cause this effect. A rather large segment of these causes involve the reflex nerve pathways, especially during surgical manipulation or traction in areas known to be sensitive to reflex changes. It is this group that we shall discuss further in more detail.

Some of the other more common causes of hypotension would include:

1. Overdosage of the pre-anesthetic medication or an unpredictable reaction, particularly in the elderly or debilitated patient.
2. The preoperative use of drugs such as cortisone and its derivatives, rauwolfia derivatives, insulin and certain phenothiazines.
3. Vasodilatation secondary to spinal anesthesia, ganglionic blocking drugs or even external heat applied to extensive areas of the body.
4. Hemorrhage and fluid loss, both before and during surgery.
5. Increased airway pressure by reduction of blood flow through the lungs.
6. Excessive depth of anesthesia.
7. Direct reaction to specific drugs or following blood transfusion.
8. Decreased CO_2 tension, which decreases the activity of the vasomotor center with resultant generalized vasodilatation and hypotension.
9. Intense pain which causes vasodilatation.
10. Synergism of drugs, e.g., thiobarbiturates and Fluothane.
11. Excessive barbiturates.
12. Hypotensive syndrome of late pregnancy with displacement of the uterus to the left and resultant occlusion of the inferior vena cava when the patient is placed in the supine position.
13. Surgical packs occluding the venous return to the heart.

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Presented at the annual meeting, Mid-South Assembly of Nurse Anesthetists, Memphis, Tennessee, February 17, 1961.

14. Myocardial infarction or intrinsic cardiac arrhythmias occurring just prior to, or during, surgery.

15. Rapid changing of the patient from one position to another or positioning which reduces the venous return to the heart.

Although reflex mechanisms may play a part in some of these other causes, we shall concern ourselves primarily at this time with specific reflexes producing a similar, and somewhat typical, overall response.

These reflexes are mediated through the vagus nerve and its interaction with other nerves by reflex activity. The distribution of the vagus nerve and its fibers is extensive and involves the head and neck, the thoracic and most of the abdominal viscera.

One of the actions of the vagus nerve is an inhibitory, or slowing, effect on the heart by reducing the rate, or pace, of the sino-auricular node in the right atrium. A complete heart block may be produced, if the vagal stimulation is sufficient, by slowing the conduction of impulses through the atria. If such a condition exists, the heart may stop momentarily or even remain in standstill[9]. Vagal stimulation, thereby, slows the heart rate. Another principal action of vagal stimulation is vasodilatation. The combination of slow heart rate and vasodilatation can produce sudden and drastic hypotension.

The clinical picture presented by patients exhibiting a reflex hypotension is usually quite consistent. The induction and early management of the anesthetic has apparently proceeded smoothly and without incident, however, these reflexes tend to occur more readily during light anesthesia and in the presence of hypoventilation, producing hypercarbia. As the site of the stimulated reflex is approached, the blood pressure suddenly drops precipitously, with a narrowing of the pulse pressure, and a bradycardia of varying degree is noted. The skin is usually found to be dry and warm. The fall in blood pressure is usually within the range of 40 to 100 mm. of mercury. The systolic-diastolic range is quite often less than 10 mm. of mercury and, in severe cases, may be completely inaudible. **An emergency situation has been produced and, unless immediate and proper evaluation and therapy is instituted, a cardiac arrest may occur.** A fall in systolic blood pressure from 120 mm. of mercury down to 80 mm. of mercury may develop without severe cerebral or renal ischemia, however, this should not be allowed to persist[11].

Preventive therapy is probably the easiest to dictate but the more difficult to achieve. Fortunately, most patients do not exhibit these severe reflex changes but in hypersensitive individuals they may occur with frightening suddenness.

Adequate premedication with atropine or scopolamine will certainly help to diminish or prevent these reflexes from occurring by blocking the vagal stimulating action of acetylcholine. Adequate ventilation of the patient is also of merit in preventing the accumulation of carbon dioxide and thus rendering the patient less susceptible to reflex changes. A finger on the pulse and frequent checking of the blood pressure will help to recognize the onset of this condition.

Corrective therapy, once the cause has been determined, might be considered in two phases; 1. Removal and correction of the cause and 2. Supportive therapy[16].

If a reflex drop in blood pressure is suspected, it should be ascertained whether or not the depth of anesthesia is sufficient. These reflexes, being more prevalent during light anesthesia, may be counteracted by increasing the anesthetic concentration.

Surgical manipulation of viscera or reflexogenic tissues is frequently found to be the cause. Surgical packs or retractors in the proximity of some of the more active plexuses may need to be removed and the surgery stopped momentarily. A dramatic response is often noted almost immediately and surgery resumed without further difficulty. In some cases, however, each repeated attempt to continue surgery in highly reflexic areas is met with the same hypotensive episode as previously encountered. Local application or injection of the area with a local anesthetic by the surgeon is sometimes necessary to block transmission of the nerve impulses. This can be accomplished satisfactorily, using anywhere from 3 to 10 ml. of 0.5% to 1% procaine or lidocaine. The weaker strength solution is preferred because it will usually prove adequate, leaving a larger margin of safety with regard to the toxic dosage of the drug. Repeat injections may need to be used if the local anesthetic begins to wear off too soon.

The intravenous administration of additional atropine or scopolamine is all that is required at times to inhibit the reflex.

Supportive therapy would include the use of a vasopressor **with which you are familiar** if the above procedures had not already corrected the situation. Trendelenburg position of the patient, needless to say, would also be helpful.

A quick check of the airway and respirations should also be made with adjustment of the method of controlled respiration or assisted respiration. Blood and fluid replacement should be started if needed, however, these reflexes are more often encountered early in the surgical procedure, before blood loss has been sufficient to alter the vital signs.

The incidence of the vagal stimulated hypotension is quite common and may be noted almost daily in the operating room. As mentioned before, the activation of this reflex occurs over a widespread area. Only a few of the more active ones will be mentioned.

In a study of 156 cases of cardiac arrest attributed to reflex depression of the heart, over half were attributed to manipulation of an endotracheal tube[9]. **Instrumentation or irritation of the pharynx, trachea or bronchi** produced by intubation, bronchoscopy, suction or sudden exposure to high concentrations of irritating gases (e.g. ether) should be done only after every precaution has been taken. Appropriate and adequate premedication, with sufficient lapse of time for distribution of the drug, should be the first step. Then a brief period of hyperventilation with 100% oxygen, immediately after intravenous induction with the thiobarbiturates and muscle relaxant, should be employed in order to ensure better oxygenation of the patient. Intubation should then be done in as short a period of time as possible with the least trauma possible. The completion of induction with irritant gases should then begin with small concentrations of the gas or vapor and the concentration gradually increased to the full induction concentration.

Direct vagal stimulation anywhere along its course and its reflex ramifications in the head, neck, chest and abdomen can cause the entity of hypotension and bradycardia. Local blocking of the nerve is usually required[5].

The **oculo-cardiac reflex** becomes evident when pressure or traction is exerted on the eye or its muscles. This reflex is mediated by way of the ophthalmic branch of the trigeminal nerve to the central vagal centers and down to the cardiac vagal nerves. Intravenous atropine or the use of a retrobulbar block by the surgeon may be necessary[4]. Studies by one investigator revealed that there were changes in cardiac rhythm associated with traction on the eye muscles in 4 out of every 5 patients[2].

The **carotid sinus reflex** is also a highly active reflex. It is noted during surgery of the neck and is parasympathetic. The sensitivity to this reflex is increased with arteriosclerotic changes, cyclopropane, morphine, thiobarbiturates and hypoxia[6].

The **celiac plexus reflex,** a sympathetic reflex, is encountered during upper abdominal surgery. This is also a highly active reflex and compression by a pack or retractor is quick to elicit it. It is more often seen during ether anesthesia. A spinal involving the lower 7 thoracic sympathetic preganglionic fibers will prevent its occurrence[6]. Relieving compression on the plexus or local infiltration by the surgeon are indicated.

The **pelvic plexus reflex** is an active reflex seen during surgery of the pelvic viscera, genitalia and perineum. Reflex laryngospasm is the predominating feature of this reflex and is usually seen during very light anesthesia. Increasing the anesthetic concentration will alleviate this response.

Traction on the esophagus or hilus of the lung also give rise to reflex hypotension. Intermittent cessation of surgical manipulation for short periods of time has been necessary for many of these cases. Although traction on the vagus is the underlying cause, infiltration of the vagus with a local anesthetic would need to be done at a much higher level than can be obtained through the surgical field. Intravenous atropine may be of some help in these cases but is not a cure. Increasing depth of anesthesia usually provides a more favorable response.

Irritation or traction of lung pleura, periosteum or peritoneum can also produce a reflex hypotension. These do not usually produce the degree of hypotension as seen above but will often show a mild, transitory effect.

CONCLUSIONS

Try to evaluate each patient before the introduction of an anesthetic, with particular regard to pre-existing blood pressure variations. Use finesse during the induction phase, with special emphasis on prevention of hypoxia and excessive manipulation within the pharynx and trachea. Close attention to the blood pressure and pulse are probably your best indicators of impending difficulty. Remain alert to the changing conditions imposed upon the patient by the surgery as well as the anesthetic. Know the effects, both desirable and undesirable, of the drugs at your disposal and know how to use them. The detection, recognition and treatment of reflex hypotension depends upon you and the skill with which this is done may mean a life, lost or saved.

(Continued on page 166)

How to Use Numorphan in Anesthesia

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The introduction of each new depressant into the world of anesthesia imposes the necessity for rapid dissemination of any accumulated observations, thus aiding others to avoid subtle pitfalls, which may render a good drug less valuable in the hands of the uninitiated. Often stereotyped ritualistic techniques must be altered slightly in order to use the new drugs to best advantage, while with imaginative alteration, a new technique evolves that may prove itself to be an additional tool of definite satisfaction to the anesthetist. The widening use of Numorphan in anesthetic procedures suggests the need to provide information based on the lessons we learned and our experience with it over the past 2 years.

TECHNIQUE OF PREMEDICATION AND INDUCTION

The use of Numorphan (oxymorphone) as the narcotic of choice before Fluothane anesthesia requires some modification of the classical techniques. The premedication dose for the average adult is 1.0 mgm., this dose being modified and individualized for the patient over 65 years of age and for the extremely ill. The addition of Nembutal to the premedication is contraindicated. In our hands, the combination of 50 mgm. of Phenergan, Numorphan, 1.0 mgm., and Scopolamine, 0.4 mgm. (all three drugs being given one hour preoperatively) has proven to be the most satisfactory. Nembutal markedly exaggerates depressive effects of Numorphan, as does a large induction quantity of Pentothal. Since Numorphan is primarily an analgesic and not a hypnotic, inclusion of a sedative to the premedicating drugs is advisable.

Assuming that the patient has had the aforementioned recommended premedication and anesthesia is about to be induced, the anesthetist must now use only the smallest amount of Pentothal. 100 to 125 mgm. (4 to 5 cc. 2½%) given quickly, has proven to be satisfactory. The mask is placed over the patient's face and ventilation is assisted as indicated, increasing the percentage of Fluothane as required to establish a level of anesthesia with this agent.

ADVANTAGES OF NUMORPHAN PREMEDICATION

In performance of short operations and procedures such as D & C, reduction of wrist fracture, cystoscopy, etc., the induction time is reduced by half, the concentration of Fluothane is reduced by 30%, and a constant level of anesthesia is much easier to maintain. Numorphan, lacking hypnotic action, allows the patient to awaken quickly and without the usual restlessness seen with Demerol—scopolamine combinations.

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Presented to the Dayton Association of Nurse Anesthetists, May, 1960.

USE OF NUMORPHAN DURING GENERAL ANESTHESIA

During surgery under general anesthesia, particularly Fluothane, the anesthetist may find it advantageous to supplement the primary agent with the addition of a narcotic analgesic. Here the potent analgesic powers of Numorphan are particularly applicable, adding to the maintenance of the patient but prolonging his awakening time. Fractional doses may be given intravenously in 0.2 mgm. increments or 1.0 mgm. may be given intramuscularly to the normal adult.

USE OF NUMORPHAN IN THE RECOVERY ROOM

The recovery room was designed primarily as the central point where the postoperative patient could be carefully observed in the immediate postoperative period. Ideally, the patient should be reacting and his normal protective reflexes present when he is returned to the recovery room. One of the many goals of the anesthetist is to get the patient awake and fully oriented. It seems rather incongruous therefore to administer in the recovery room an analgesic such as Demerol with its strong and undesirable side effect of hypnosis. Numorphan in 0.75 to 1.0 mg. dosage, repeated in fractional doses, as necessary, furnishes analgesia without precipitating the patient back into a deep sleep. The use of oxymorphone narcotic as a premedicant and as an adjunct to light general anesthesia, and then in the recovery room contributes to the achievement of this goal of wakefulness and complete orientation and maintains a high degree of analgetic comfort.

SUMMARY

Use of Numorphan (oxymorphone) for premedication contributes to the ease of induction and maintenance especially when using Fluothane. For additional preoperative sedation, we used Phenergan. Because it accentuates respiratory depression, Nembutal should not be used in combination with Numorphan. If Pentothal is used for induction, the total amount should not exceed 125 mgm.

The goal of analgesia without sleep in the recovery room is best served by administering fractional doses of Numorphan as required and by avoiding the use of narcotics which possess strong hypnotic properties.

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Triflupromazine Hydrochloride† As An Adjuvant to Surgical Anesthesia

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Modern techniques in surgical anesthesia are designed to provide anesthesia of the depth required for the scheduled operation with a maximum of safety and minimum of discomfort for the patient. An integral and routine part of present-day procedure is to administer adjunctive drugs to the patient prior to anesthesia to allay anxiety, to reduce resistance to anesthesia and to counteract unwanted effects from the subsequent administration of the anesthetic drugs[1]. The medicaments usually given before anesthesia include a sedative or hypnotic agent together with a drug to reduce excessive salivation and mucosal secretion[1]. Since the usefulness of chlorpromazine in presurgical anesthesia was discovered,[2, 3] a number of other phenothiazine derivatives, subsequently introduced, have proved useful as preanesthetic medicaments because of their tranquilizing effect, their antiemetic action and their potentiating effect on anesthetic and hypnotic agents so that smaller amounts of these agents are effective.[4, 6] Among the phenothiazine compounds that have been used as adjuvants to surgical anesthesia, triflupromazine has been found to be especially helpful not only in relaxing and calming the patient with an accompanying diminished reaction to pain[7, 8] but also in preventing or treating postoperative nausea and vomiting.[6, 9]

The present report concerns the use of triflupromazine hydrochloride routinely administered before anesthesia to patients at St. Francis Hospital, La Crosse, who were scheduled for surgery for a variety of conditions. As given, triflupromazine had an excellent tranquilizing effect in the majority of patients without serious depression of the cardiovascular and respiratory systems. The antiemetic action of the drug was one of its superior assets, especially when used with the anesthetic agents administered to the patients included in this report. A brief account of our experience with triflupromazine hydrochloride as a premedicating agent for surgical anesthesia follows.

PROCEDURE

A total of 70 surgical patients received triflupromazine hydrochloride, usually before anesthesia, but in a few cases postoperatively. Fifteen of the 70 patients were 15 years of age or younger, the youngest being 4 years old. The range in age of the other patients in the series was from

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*St. Francis Hospital, La Crosse, Wisconsin.
†Supplied as Vesprin by E. R. Squibb & Sons.

17 to 76 years. Twenty-two of the 70 patients were male, and 48 were female. The conditions found in these patients which required operative procedures are listed in table 1.

Established procedure was followed in preparing these patients for anesthesia except that, in addition to the usual premedication, triflupromazine was administered about one-half hour later, approximately one hour before the scheduled arrival of the patient in the operating room. Premedication in the majority of cases consisted of meperidine (Demerol) and either atropine or scopolamine. Triflupromazine was administered intramuscularly in every case except one who received the drug intravenously during surgery. All patients except one received the drug preoperatively; one patient was given triflupromazine immediately after surgery. The usual dose of triflupromazine was a single injection of 10 mg. of the drug although smaller single doses were given to most of the children and larger single doses were administered in one or two patients. In a few instances, injections of triflupromazine were repeated once, twice, or even four times.

Table 1.

Condition	**Number of Patients**
Hernia, inguinal or ventral	6
Cholecystitis	10
Appendicitis	7
Breast tumor	5
Uterine disorders and other gynecologic conditions	9
Inflammation or lesion of the lung	2
Cancer of the colon	2
Ruptured disc or bone fracture	5
Goiter	2
Baker's cyst, varicose veins and other conditions requiring minor surgery	5
Chronic tonsillitis	4
Multiple caries requiring exodontia	2
Strabismus and other disorders of the eye requiring corrective surgery	11

Details of dosage and administration of triflupromazine as well as of the other medications administered as premedicating agents for the various operating procedures scheduled in the different patients in this series are shown in Table 2. The effects observed in the patients following such treatment also appear in Table 2.

RESULTS

Good tranquilization was observed in 53 and fair tranquilization in 15 of the patients treated preoperatively with triflupromazine prior to anesthesia. Little or no tranquilization was apparent in one child of 5 years scheduled for tonsillectomy who was extremely frightened and nervous. The single patient who received triflupromazine only after operation showed only a fair response to the drug. A patient was recorded as showing good tranquilization if on arrival in the operating room he was calm, relaxed and free from apparent apprehension over the impending operation. Some were alert, some were drowsy, but all could be aroused to follow the instructions of the anesthetist. The tranquilizing effects of the administered agents were considered to be only fair if the patient

Table 2. Summary of Results with Triflupromazine Hydrochloride As an Adjunct to Surgical Anesthesia[1]

Surgical	No. of	Age	Sex		Preanesthetic and/or Postanesthetic Medication[2]				
Procedure	Cases	(years)	M	F	Medicament	Amount	When Given	No. of Patients	Results
Herniorrhaphy	6	29 to 71	4	2	Meperidine HCl and	50 mg.	Preop.	6	Good tranquilization in all with patients arriving in operating room calm, relaxed and cooperative. No emesis in any patient. Postoperative nausea in 1 patient controlled by a single injection of triflupromazine.
					Atropine SO_4	1/150 gr.			
					Triflupromazine HCl	10 mg.	Preop.	6	
					Triflupromazine HCl	10 mg.	Postop.	1	
Cholecystectomy	10	29 to 67	1	9	Meperidine HCl and	50 to 75 mg.	Preop.	8	Good tranquilization in 6 cases, only fair in 4 with the patients arriving in the operating room calm but apprehensive. No emesis in 6 but 3 patients vomited immediately after recovery and 1 complained of nausea. Hypotension developed in 2 patients during surgery (B.P. of 84/60 and 90/60). Both responded satisfactorily to intravenous mephetermine (Wyamine) and phenylephrine (Neosynephrine) drip for 10 minutes. Fair results in the single patient treated postoperatively with triflupromazine.
					Atropine SO_4	1/150 gr.			
					Triflupromazine HCl	10 mg.	Preop.	7	
					Triflupromazine HCl	10 mg.	immediately postop.	1	
Appendectomy	7	4 to 76	5	2	Meperidine HCl and	50 to 75 mg.	Preop.	6	Good tranquilization in 5, fair in 2. One episode postoperative vomiting in 1 despite treatment with triflupromazine but no vomiting after operation only nausea. Postoperative nausea in 2 and vomiting in 1 patient but these symptoms soon subsided after treatment with triflupromazine.
					Atropine SO_4	1/150 gr.		7	
					Morphine SO_4	1/6 gr.		1	
					Triflupromazine HCl	4 mg.	Preop.	1	
					Triflupromazine HCl	10 mg.	Preop.	2	
					Triflupromazine HCl	10 mg.	a 6 hr. x 2 preop.	1	
					Triflupromazine HCl	10 mg.	a 3 to 4 hr. x 4 postop.	1	
					Triflupromazine HCl	10 mg.	12 hr. postop.	1	
					Triflupromazine HCl	10 mg.	immediately postop.	1	

(table continued)

Breast tumor	5	19 to 61	0	5	Meperidine HCl and Atropine SO_4	50 to 75 mg. 1/150 gr.	Preop.	5	Good tranquilization in 3 cases, only fair in 2. No emesis in any. Apnea developed in the 1 patient who received 20 mg. of triflupromazine but this was relieved in 5 to 10 min. by the administration of methylglutarimide (Megimide). Blood pressures dropped in 3 cases but this was transient and no vasopressor was given to any of the 3.
					Triflupromazine HCl	10 mg.	Preop.	4	
					Triflupromazine HCl	20 mg.	Preop.	1	
Gynecologic Surgery (Hysterectomy—4 Ovarian cyst—1 Cystocele repair—1 D&C—2 C-Section—1)	9	22 to 56	0	9	Meperidine HCl and Atropine SO_4	50 to 100 mg. 1/150 gr.	Preop.	4	Good tranquilization in 8 cases, only fair tranquilization in 1. No nausea, no emesis in any case.
					Morphine SO_4 and Scopolamine	1/6 gr. 1/150 gr.	Preop.	1	
					Amobarbital and Scopolamine	3 gr. 1/150 gr.	Preop.	1	
					Atropine SO_4	1/150 gr.	Preop.	1	
					Triflupromazine HCl	10 mg. IM	Preop.	7	
					Triflupromazine HCl	10 mg. IV	during surgery	1	
					Triflupromazine HCl	10 mg. IM	Postop.	1	
Thoracotomy	2	17 to 56	1	1	Meperidine HCl	50 mg.	Preop.	1	Fair tranquilization in both cases. On arrival in operating room the patients were calm but slightly apprehensive though they did not seem to be fearful. No respiratory depression and cough reflex was present after surgery in both. No emesis in either. Nausea in 1 for 1 hr. postoperatively.
					Meperidine HCl and Atropine SO_4	25 to 75 mg. 1/150 gr.	Preop.	2	
					Triflupromazine HCl	10 mg.	Preop.	1	
					Triflupromazine HCl	20 mg.	Preop.	1	
					Triflupromazine HCl	10 mg.	Postop.	1	
Laparotomy	2		0	2	Meperidine HCl and Atropine SO_4	50 to 100 mg. 1/150 gr.	Preop.	2	Good tranquilization in both cases with relief of preoperative nausea and vomiting. No postoperative vomiting. Nausea 24 hrs. in 1 case after surgery relieved by 10 mg. of triflupromazine intramuscularly given.
					Triflupromazine HCl	10 mg.	Preop.	1	
					Triflupromazine HCl	10 mg.	q. 6 hr. x 6 preop.	1	
					Triflupromazine HCl	10 mg.	once postop.	1	
Orthopedic Surgery (Laminectomy—3 Open reduction fracture rt. scapula—1 Closed reduction and cast for fracture fibula—1)	5	40 to 65	2	3	Meperidine HCI and Atropine SO_4	50 to 75 mg. 1/150 gr.	Preop.	5	Good tranquilization in 3 cases, only fair in 2 with both patients awake and apprehensive on arrival in the operating room. No emesis in any patient, slight nausea in 2. Blood pressure dropped during surgery in 1 patient from 122/70 to 120/50 but rose to 130/80 postoperatively without vasopressor drugs.
					Triflupromazine HCl	10 mg.	Preop.	5	

Thyroidectomy	2	40 to 52	0	2	Meperidine HCl	50 mg.	Preop.	2	Good tranquilization in both cases, no emesis, no nausea.
					and				
					Atropine SO$_4$	1/150 gr.			
					Triflupromazine HCl	10 mg.	Preop.	1	
					Triflupromazine HCl	20 mg.	Preop.	1	
Minor Surgery	5	6 to 49	2	3	Meperidine HCl	50 mg.	Preop.	4	Good tranquilization in 4 cases, only fair in 1. No emesis nor nausea in any.
					and				
					Atropine SO$_4$	1/150 gr.			
					Triflupromazine HCl	10 mg.	Preop.	4	
					Triflupromazine HCl	10 mg.	Postop.	1	
Tonsillectomy	4	5 to 15	1	3	Meperidine HCl	25 to 50 mg.	Preop.	3	Good tranquilization in 3 cases, but poor results in 1 with the child (5 years) extremely apprehensive and uncooperative on arrival in the operating room. No emesis, no nausea in any case.
					and				
					Scopolamine	1/150 gr.		4	
					Triflupromazine HCl	5, 6, 10, 12 mg.	Preop.	4	
Exodontia	2	37 to 59	0	2	Meperidine HCl	50 mg.	Preop.	2	Good tranquilization in both; no emesis, some postoperative nausea in 1.
					and				
					Scopolamine	1/150 gr.	Preop.	1	
					or				
					Atropine SO$_4$			1	
					Triflupromazine HCl	10 mg.	Preop.	2	
Eye Surgery (Strabismus—9 Enucleations—2)	11	5 to 55	6	5	Meperidine HCl	25 to 75 mg.	Preop.	9	Good tranquilization in all but 2 patients. A 6-year-old boy with esotropia and a 52-year-old woman scheduled for enucleation were quiet but apprehensive on arrival in the operating room. Postoperative emesis in 2 patients and nausea in 1 relieved by triflupromazine.
					Scopolamine	1/200 to 1/150 gr.	Preop.	8	
					Atropine SO$_4$	1/200 gr.	Preop.	1	
					Thiopental Na	1 Gm (1cc.) rectally	Preop.	1	
					Triflupromazine HCl	4, 5, 7 or 10 mg.	Preop.	10	
					Triflupromazine HCl	10 mg.	Postop.	1	
					Triflupromazine HCl	10 mg.	q. 8 hrs. x 3 Postop.	1	

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[1] Thiopental Na (Pentothal) and nitrous oxide in the majority of cases
Thiopental Na, nitrous oxide and bromochlorotrifluorethane (Fluothane) in several cases
Vinyl ether (vinethene) and ethyl ether in a few cases

[2] Administered intramuscularly unless otherwise stated
The sedative and analgesic agents were usually administered 1.5 hr. preoperatively
Triflupromazine was usually administered 1 hr. preoperatively unless otherwise stated.

arrived in the operating room calm but apprehensive; in a few instances these patients displayed blood pressure levels above their previous readings. Ten patients complained of nausea after surgery and 8 actually vomited, but these symptoms soon subsided spontaneously or were relieved after the administration of one or two injections of triflupromazine. Blood pressures dropped in six patients during anesthesia but in only 2 of the 6 were vasopressor drugs administered. Both patients responded satisfactorily to these agents. Apnea developed in one patient who received a single injection of 20 mg. of triflupromazine prior to anesthesia but this was relieved in 5 to 10 minutes by the administration of methyl glutaremide (Megimide). No serious depression of circulation or respiration was seen in any patient. No other untoward effects were seen.

CONCLUSIONS

The observations recorded in this group of 70 surgical patients treated with triflupromazine hydrochloride as a premedicating agent indicate that it is a valuable adjunct to surgical anesthesia. The procedure in which triflupromazine hydrochloride was given in addition to usual preanesthetic medication provided "good tranquilization" in the majority of cases and excellent control of postoperative nausea and vomiting.

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Some Considerations of Anesthesia for the Patient with Cardiac Disease

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The great progress that has been made in the field of anesthesia during the past few years has opened many doors for the surgical treatment of patients who previously would have been allowed to suffer and probably expire as a natural course of events. Among the many pathological conditions which often led to the classification of a poor surgical risk was the patient with cardiac disease. This still can be said if we continue to practice anesthesia the way we did ten or fifteen years ago. The majority of us in this field prefer to stay abreast with the times and accept newer methods and drugs as they are offered by the various research organizations in an effort to render a better service to the individual patient. No offense is intended nor any reproach cast upon the older methods of anesthesia or those who still use them. Their methods may be better for the patient than some of our more recent methods — the test of time and mortality will prove which is superior.

HEART LESIONS

The specific type of cardiac disease is less important than the effective function of the heart. A simple classification of patients into those with congenital and those with acquired lesions will perhaps be helpful. Congenital lesions are a defective problem and will not be considered at this time. Acquired lesions are seen and exposed to our various anesthetics more frequently, and should be considered from a practical approach[1].

Acquired lesions or disease tend to affect both the myocardium and the valves so that, in the earliest stages, the heart muscle is weakened. In normal circumstances, the reserve of the myocardium is so great that gross abuse can usually be tolerated provided it is not unduly prolonged. In the early stages of heart disease this reserve, even though reduced, is adequate to sustain the patient through an average operation and anesthetic. Should failure occur, it is most likely to be in the heart where an acquired lesion is present and where the myocardium is not only less tolerant of such stress but more susceptible to arrhythmia production. These facts apply equally whether an operation is performed upon the heart or the great vessels directly associated with it, or upon some other part of the body. The whole body of the patient must still be considered, not only the part containing a surgical disease.

In the clinical practice it is the cardiac reserve that must ultimately be considered by the anesthetist when deciding whether a patient is suitable for an anesthetic—and also surgery. This great decision should be made only after a specific detailed investigation.

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Presented to the Nebraska Association of Nurse Anesthetists, Omaha, Nebraska, October 20, 1960.

Once the patient is presented for anesthesia, it is well to confer with the previous physician after carefully checking the record and all allied information available. This is done to concur with the physician and thus offer a better type of anesthesia to the patient. In the evaluation of the patient it is good to know the ability of the heart at rest and under stress, the most tolerable position for the patient, and any anesthetic problems previously affecting the patient. Without an adequate amount of information it is impossible to state emphatically that the patient will be unable to tolerate an anesthetic or tolerate one without side effects. A specific anesthetic for this type patient might be fatal but none of us wish to be accused of violating the Fifth Commandment, just because we were too lazy to investigate the patient's history or too proud to think that we could always bring every patient through the anesthesia. The administration of an anesthetic to a patient with cardiac disease is not easy and should never be instituted without careful consideration and planning.[2, 3]

With proper care most patients with cardiac disease tolerate both anesthesia and surgery. Some die because of major or even minor errors of technic and judgment, and others from natural causes — too little cardiac reserve to tolerate the insult of the drugs and manipulations.

It is well established that, although both anesthesia and surgery lead to specific alterations in the hemodynamics of the heart and circulatory system, the tolerance of this system is considerable in a healthy patient. In the presence of disease, minor upsets become accentuated and occasionally lead to cardiac or circulatory failure or even death. What are the little upsets that might be prevented and how can we be better prepared for their appearance?

Any alteration in the vital signs which is not expected may be referred to as an upset. Little upsets can well enlarge themselves into a major disaster. Potential upsets can be detected by observing the patient, reviewing the history, evaluating the blood pressure in various positions (especially the one to be used in surgery), estimating the pulse pressure variations, pulse rate and quality, or any degree of peripheral cyanosis and vasoconstriction. By this means it can be ascertained whether the patient has a low cardiac output and is in need of close attention[4]. A rapid weak pulse, indicating some degree of cardiac failure and potential pulmonary edema, breath odor and skin texture — all aid in selecting the proper anesthetic.

PREOPERATIVE MEDICATION

The actual selection of the type of anesthetic will depend on many factors and this selection should be made only after due consultation by the internist, surgeon and anesthetist[5]. Unfortunately, many of our colleagues on such a team do not know all the possibilities and reactions of all anesthetics, and this is the time for those experienced in anesthesia to offer good concrete information. This can be done with diplomacy. This is not a time for personal pride to become illuminated, but rather humility and a sincere desire to seek the safest method of serving the patient.

The big decision is whether to use regional or general anesthesia—which would be safer for the patient, meet the surgical requirements and cause the least discomfort? If the anesthesia can be conducted in such a way that the patient will deviate only slightly from his current normal physiological pattern, then a superior job will be done.[2, 3]

The questions arise — when does the anesthesia start and when should be the proper time for the anesthetist to take an active part? The onset of anesthesia actually starts when the patient is first informed of the pending surgery. At this time the anesthetist must begin to consider the many possibilities within his field as applied to this particular patient in this current situation. The unlimited value of visiting the patient prior to "D" day and "H" hour cannot be forgotten. This is an excellent method of sedation and comfort for the patient —someone is taking a personal interest—along with offering us the opportunity of gaining valuable information from our observations and inspections. Premedication is important and should consist of only that actually needed for the patient's safety[6]. The immoral routine use of standard doses of sedatives, opiates, belladonna drugs, tranquilizers, etc., is mentioned only to be condemned. Frequently, patients given only a mild antihistamine for sedation, or perhaps nothing, do much better during the anesthetic period and surgery than those who have been "drugged" by premedication. Selection of the drugs for premedication should be given as much thought as the actual anesthetic. Only enough sedative should be given to provide a mild to moderate degree of mental sedation.

Mental sedation is of special importance for patients with cardiac disease as the stress of fear can be sufficient to induce an anginal attack or precipitate the acute onset of pulmonary edema. Complete mental sedation may appear nice to the uneducated in anesthesia, but at what price is it obtained? Usually, when it is truly complete there is a grave insult to the cardiac and respiratory centers of the body along with the production of rather sluggish activity by the vital organs which we attempt to keep fairly close to a normal physiological state.

Only minute doses of belladonna drugs are usually required to produce dryness of the respiratory tract; an overdose will produce a rapid heart rate and may lead to heart failure[7].

The production of a peaceful state of basal narcosis with barbiturates is unsuitable for the patient with cardiac disease, since it leads to circulatory depression and marked restlessness in the immediate post-anesthetic period when surgical pain is first perceived.

When the patient is brought into the surgical area it is extremely important to determine the effect of the premedication. If it has been ineffective or has made the patient markedly depressed, surgery may have to be postponed rather than endanger the patient's life. Another indication for postponement at this stage is a marked deterioration in the patient's condition such as the onset of incipient or acute pulmonary edema[8].

ANESTHESIA INDUCTION

The next step is the induction of the true anesthesia. In regional anesthesia the physician assumes the responsibility and must have all the assistance available, while in general anesthesia the anesthetist assumes command[8].

Regional anesthesia includes local infiltration, nerve blocks, ganglion blocks, epidural and spinal technics. Refrigeration types of anesthesia may be considered with this group if only a part of the body is to be anesthetized. Should any of these be used, it is our job to insure an adequate blood pressure, fluid replacement, possible sedation and in general watch over the patient very carefully. The patient under regional anesthesia is entitled to our care just as much as the patient under a general anesthesia. (At the last statistical evaluation of mortality it was quite evident that not all people die in their sleep.)

Poor handling of the induction and establishment of a general anesthesia can mar the subsequent success of the surgical procedure. Quite often a patient is less disturbed by the expectancy of going to sleep with an injection than with a mask over his face. Nevertheless, the latter may be necessary either because pentothal and similar hypnotic drugs are contraindicated, or because oxygen must be administered. The dangers of pentothal in cardiac disease are at their greatest in low cardiac output states such as mitral stenosis and constrictive pericarditis[1]. It is suggested that when pentothal is contemplated for induction, a test dose of 25 to 50 mg. be given and the patient observed for any possible allergic reaction or change in the vital signs. It is well to remember that circulation time in cardiac disease is quite often slowed down and the distribution of the drug delayed[7]. It should be explained to the patient that oxygen will be given while he is going to sleep thus avoiding the anxiety when a mask is placed over his face. Once our test dose has been observed in action, a minimal induction dose may be given the patient receiving oxygen by mask. As the patient goes to sleep oxygen is continued and preparation made to insert an airway. There is probably a minimal effective dose of pentothal that most patients can tolerate; but, when assessment of the patient's reserve is difficult, this and similar hypnotic agents are better avoided and induction carried out with cyclopropane and oxygen. The technic used prior to the clinical introduction to pentothal should not be forgotten i.e., the lost art of gas induction which can and should be pleasant for both the patient and the anesthetist.

Many people will avoid the use of intravenous hypnotics and thus have the patient more apt to recover full consciousness immediately at the end of the surgical procedures. The use of nitrous oxide may be made but the danger of hypoxia is always present, dependent on the skill of the anesthetist. Any feeling of suffocation occurring during the prolonged induction may lead to some apprehension and tachycardia. The induction with nitrous oxide can be shortened considerably if the patient is denitrogenated prior to induction by merely breathing 100% oxygen. Then when nitrous oxide is given, it will be picked up rapidly and induction will be shortened. If this type induction or ethylene-oxygen mixture is used, intubation of a patient should never be attempted without benefit of a muscle relaxant.

The use of Fluothane-nitrous oxide-oxygen for an induction may be most pleasant for the patient and the anesthetist. This type of induction may be carried down to intubation without muscle relaxants and with little difficulty, although it requires special attention and alertness of the anesthetist.

After the patient has been fully oxygenated, intubation is carefully performed with Anectine or some other muscle relaxant, as soon as unconsciousness is established. The patient is then allowed to recover, but not allowed to wake up, (this would be fatal, especially to a patient with cardiac disease) from this test dose of muscle relaxant and re-establish his own respiratory pattern prior to the administration of any other relaxant.

A good method of intubation is a topical anesthetic such as cocaine 5% used as a spray to the superior and inferior aspects of the vocal cords and larynx prior to the insertion of a well lubricated, cuffed endotracheal tube. An intubation that is protracted or causes strain is potentially dangerous and may be fatal. An adequate dose of the muscle relaxant for intubation will assist in preventing these complications and allow for the effect of a topical anesthetic to become established.

If total paralysis is to be accomplished, the same respiratory pattern established by the patient should be attempted as far as possible. The breathing pattern of the patient should not be changed. An adequate amount of muscle relaxant should be given for smooth controlled respirations, which must be constantly observed for adequate depth. Hyperventilation may appear to be very good for the patient from a standpoint of oxygen availability. The respiratory alkalosis and metabolic acidosis which follow this type of ventilation may be more of a shock than a patient with cardiac disease can tolerate initially or during recovery[9].

When a mild degree of muscle relaxation is desired, only minimal doses of relaxants should be given intermittently and respirations assisted but not controlled. Lapse of active respirations by the patient is the loss of a very important sign indicating the depth of anesthesia. A very small amount of a muscle relaxant drug is usually sufficient for good relaxation during most procedures, and it is inadvisable to over-relax the patient especially one with cardiac disease.

ANESTHESIA MAINTENANCE

The maintenance of anesthesia is not an easy or minimal task. Giving undivided attention to a patient, who is under constant anesthesia, should give the anesthetist a significant degree of work satisfaction[10]. Previous arrangements should be made for additional help or supplies in case they are needed, thus avoiding the neglect of the patient and possibly a disaster during anesthesia. When other drugs are needed they should be administered cautiously especially those given intravenously, as these cannot be reclaimed and must be eliminated or destroyed in the body. The use of supplemental drugs should be avoided whenever possible, as the least number of agents that enter into any anesthesia, or medical treatment, will make for the least number of complications. Balanced anesthesia is very good provided it does not produce adverse reactions.

In maintaining the anesthesia, the equilibrium of the cardiovascular system is of great importance, especially one with a diseased heart. This equilibrium is best maintained by constant vigilance over the patient[6]. The use of various electronic apparatus is very nice and often quite informative, however, a sensitive finger continually monitoring the pulse should never be underrated.

The problem of hypotension will most likely present itself while the patient is on the operating table. This condition can be prevented by means of effective ventilation, replacement of blood loss, and the greatest possible patience and delicate handling by the surgeon. Intelligent anticipation of hypotension periods with various surgical maneuvers will insure against the unwarranted use of vasopressor drugs.

Vasopressors are used only when the systemic systolic pressure falls below 60 mm. mercury and fails to respond to a rest period. In such a case it is assumed that the blood loss has been replaced, but it is worth noting that the response to blood transfusion following even slight hemorrhage may be delayed 10 to 15 minutes in the patient with cardiac distress. Methedrine or Vasoxyl given intravenously are two of many useful myocardial stimulants. However, the sudden stimulation of the patient, and the potential hypotension which usually follows the initial rise in blood pressure should be considered. The various methods of altering the position of the patient, although inconvenient to the surgical team, may correct the hypotension quite adequately and insure greater safety to tne patient[1].

The variety of cardiac arrhythmias is often quite alarming to the anesthetist and it must be remembered that most of these occurring during an operation are transient and caused by direct stimulation to the heart or major vessels. A normal or pre-existing rhythm can generally be expected to return on removal of the causative factor. The less frequent causes of arrhythmias, such as hypoxia or hypercapnia, excessive depression by drugs, etc., should not be overlooked[6]. General deterioration of the cardiovascular system cannot be corrected by anesthesia. If we consider the presence of arrhythmias as the measure of the insults leveled against the heart, then the correction and future prevention of these arrhythmias may be initiated.

It is imperative that only the fluid actually lost should be replaced; as overloading a tired and crippled heart and its vascular system is serious and can produce congestive failure. Equally serious is the lack of fluid replacement[2].

If pulmonary edema presents itself during surgery, it is better to continue the surgery and rapidly relieve the cause of the failure than to alleviate it by medical means alone. Some beneficial procedures are: 1. Anti-Trendelenburg position to ease the pulmonary congestion (may be combined with venous tourniquets applied to the lower limbs). 2. Efficient suction to remove the edema fluid from the lungs and to enable better oxygenation. 3. Possible venesection—however, most patients will have lost too much blood during surgery through the peripheral vasodilation of general anesthesia. 4. Removal of the cause if possible (chemical irritation of the pulmonary epithelium, or oxygen poisoning, etc.)[1].

At the termination of the operation, the patient should be returned to his pre-anesthetic state as rapidly and safely as possible. To prolong the transition from anesthesia to a stage of circulatory and respiratory equilibrium is potentially very dangerous. Ineffective ventilation is probably the commonest single cause of difficulty[9]. This does not necessarily imply residual curarization, but is often the combination of tiredness, wound pain and pulmonary congestion. When the endotracheal tube can no longer be tolerated, an element of upper respiratory resistance may be present if the patient is too weak to keep his tongue forward.

Full oxygenation must be insured, if necessary with the use of a mask. The position of the patient depends on the level of consciousness, the systolic blood pressure, and the presence or absence of pulmonary congestion. Fully conscious patients of this type are happiest sitting up, but this position must not be completely assumed until the systolic pressure is at least 100 mm. mercury. Usually a gradual change from the supine to the lateral position is best and then with a slow elevation of the head of the bed as tolerated — indicated by close observation of the vital signs. A return to the supine or semisitting position is then usually acceptable to the patient.

Analgesics should not be given to the patient until he has fully recovered to a conscious level and has become settled in bed. Although persistent pain increases the patient's exhaustion, the too early injudicious use of analgesics can be of greater danger.

Recovery from unconsciousness may be delayed unless the bare minimum of narcotics has been used. Patients with severe heart disease are often so completely exhausted at this state that anesthesia may emerge into deep sleep and the patient can only be aroused with difficulty. If the elimination of the anesthetic can be assumed, yet coma remains, then other causes for it must be investigated.

The anesthetist must remain in the recovery area and support the patient until he is fully awake and stable—then only are the anesthetist's responsibilities completed.

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Patients With Cardiac Disease:

Treatment and Preparation for Major Surgery

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An internist is frequently asked to see a patient with cardiac disease who is to undergo a major surgical procedure. No one, regardless of age, should be denied this type surgery unless there are absolute contraindications. Even then, in emergencies, the contraindications may have to be waived in an attempt to prolong life. These patients tax the skills and ingenuity of the internist, surgeon and anesthetist to the fullest extent. However, in elective surgical cases, time is available to decrease operative risk, thus reducing postoperative complications.

PREOPERATIVE PERIOD

The first requisite is a detailed history and a complete physical examination, which may reveal major system diseases. Baseline laboratory studies should be performed and these include: hemogram, urinalysis, chest roentgenogram, electrocardiogram, serum electrolytes, blood grouping and Rh factor, bleeding, coagulation and prothrombin times. A chest fluoroscopy, venous pressure, circulation time, blood volume determination and pulmonary function studies may also be indicated.[8, 9, 10] A complete assay of the patient's cardiac function is basic in determining operability[11].

As older patients with cardiac disease have lower resistance to infections, it is mandatory that antibiotics be given preoperatively to eradicate or control these infections.[3, 4, 5, 8] This is especially true in chronic bronchopulmonary disease with superimposed acute infection.

Good medical management before surgery can aid in improving malnutrition, avitaminosis, anemia, hypoproteinemia, endocrine disorders, polycythemia, blood dyscrasia and a host of other conditions. When patients are on long-term steroid therapy for a pre-existing disease, special attention should be given to augment and regulate the dosage during the operative and postoperative periods.[8, 11] Blood volume deficits must be corrected by adequate blood transfusions prior to, during, and after surgery in order to promote optimum surgical tolerance[3]. Fluid and electrolyte imbalances should also be corrected.[2, 3, 4, 7, 8, 9, 11]

Cardiovascular diseases increase surgical morbidity and mortality.[7, 8, 9] The diseased heart is unlikely to tolerate hypoxia, hypercapnia, anemia, hypotension, deep anesthesia and reflex phenomena. [5, 11] Especially hazardous are uncontrolled arrhythmias, severe coronary insufficiency, certain types of heart block, aortic valvular disease, myocardial failure, active myocarditis, endocarditis or pericarditis, recent myocardial infarction, hypertension with paroxysmal nocturnal dyspnea, and chronic cor pulmonale. It must be realized that patients with heart disease are more susceptible to postoperative renal, respiratory, and vascular complications, and that other disease states may contribute to or actually precipitate congestive heart failure.[8, 9, 11]

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Presented to the Nebraska Association of Nurse Anesthetists, Omaha, Nebraska, October 20, 1960.

Congestive heart failure is treated by rest, salt restriction, digitalization, diuretics, and oral potassium chloride when indicated[8]. Potassium deficiency causes patients with cardiac disease to be more prone to digitalis toxicity.

Hypertension is best controlled by antihypertensive agents, preferably the Rauwolfia derivatives[8]. These drugs are known to produce significant bradycardia and hypotension during anesthesia and are preferably to be discontinued prior to surgery[11]. Patients with paroxysmal nocturnal dyspnea should have, in addition to 2 to 3 weeks of bed rest, digitalis and a trial of diuretics.[8, 11]

The condition of patients suffering from coronary insufficiency with angina pectoris may improve after the administration of oxygen, nitroglycerin and long acting vasodilator drugs[8].

When Adams-Stokes syncope is present, the patient should be given 0.5 to 1.0 mg. atropine sulfate every 8 hours and ephedrine sulfate 50 to 100 mg. every 4 to 6 hours, or isopropylnorepinephrine (Isuprel) one 15 mg. glosset sublingually every 3 to 4 hours[8]. He should be digitalized if cardiac failure is also present.

Any arrhythmia should be brought under control and warrants immediate attention if it occurs during the operative or postoperative period. Auricular fibrillation or flutter, auricular or nodal tachycardia, unifocal or multifocal auricular or nodal premature beats respond best to digitalis or quinidine, or both. Auricular tachycardia with A-V block due to digitalis toxicity is treated best by discontinuing digitalis and giving potassium orally or by intravenous infusion. Unifocal or multifocal extrasystoles frequently respond to potassium administration. Ventricular tachycardia is particularly hazardous and in this instance pronestyl (procaine amide) is the drug of choice when given intravenously at a rate of 200 mg. per minute.[6, 8, 9] A continuous electrocardiogram should be made and the drug discontinued as soon as the arrythmia is interrupted.[8, 11]

Patients having had acute myocardial infarction should have surgery postponed until 3 months after the infarction has stabilized. Those with evidence of myocarditis, pericarditis or subacute bacterial endocarditis should not undergo surgery until all evidence of active infection has subsided[8].

Patients with cardiac disease and having thyrotoxicosis should be treated with thiouracil drugs or radioactive iodine or both as indicated. They should be made as euthyroid as possible before surgery. Similarly other endocrine disorders such as diabetes mellitus, adrenal insufficiency, myxedema, etc., should be controlled by appropriate management.

Improvement may be obtained in patients with chronic cor pulmonale due to chronic bronchial disease with associated pulmonary fibrosis or emphysema by treatment of chronic and superimposed acute bronchial infections which will usually respond to appropriate antibiotics. Sputum liquefacients, nebulized bronchodilators with intermittent positive pressure and oxygen will facilitate expectoration of viscid secretions and alleviate any existing bronchospasm and cyanosis. Patients with bronchiectasis may be additionally improved by postural drainage. When the hematocrit exceeds 53% a phlebotomy is indicated. Digitalization should be undertaken if myocardial failure supervenes[8]. When oxygen is given for cyanosis great care should be taken to avoid depression of the medullary center of respiration and subsequent respiratory failure.[4, 8] Oxygen should be used in extreme emergencies only, and then for short intervals at a flow rate of 2 to 3 liters per minute. The use of narcotics is hazardous in these patients and may cause anoxia by depressing the respiratory stimulus.

OPERATIVE PERIOD

The surgeon and anesthetist should be constantly on the alert for vital signs during the operative procedure, especially in patients with arrhythmia. An electrocardiogram monitor is invaluable in the early detection of serious arrhythmia or cardiac arrest during surgery[11]. Except in severe pulmonary insufficiency, there should be a high concentration of oxygen at all times. The airway must be kept patent. Any fluid or blood loss must be replaced immediately to avoid hypotension and shock with its associated cerebral, cardiac or vascular hazards[8]. Excessive administration of fluid or blood can precipitate pulmonary edema especially in cardiac patients.

Special consideration should be given to anesthetic management. A rather wide variety of anesthetic agents and adjuvant drugs are available[2]. With proper premedication and appropriate anesthesia in the hands of a skilled anesthetist or anesthesiologist the surgical risk, and operative and postoperative complications are further minimized.

The physician must always bear in mind the anxiety the patient and his family are undergoing. It is his duty to explain to the family, in simple, understandable language, the patient's condition, the contemplated surgical procedure, and the prognosis. It is important that the physician gain the confidence of the patient and his family to become their friend and advisor.[1, 8]

SUMMARY

A patient with cardiac disease who is to undergo major surgery should be thoroughly examined and evaluated regarding this disease or any other disease state which may be present. Frequently much can be done to improve a poor risk patient with heart disease to an acceptable surgical risk. Elective surgery is best deferred until the patient is brought into optimum condition.

A poor risk patient needing emergency surgery requires the combined skills of the attending physician and/or internist, the surgeon and anesthetist or anesthesiologist. Postponement of surgery may not be possible for very long and major contraindications as for elective surgery may have to be waived. Even in these instances, with the short time available, emergent preoperative management will often improve chances for survival and reduce postoperative morbidity and complications.

(Continued on page 166)

Hospital Safety

Harriet L. Aberg, C.R.N.A.

Safety Displays

It is gratifying to note that some of our members are active in the over all safety programs of their hospitals. Safety is difficult to sell sometimes, although the safe worker suffers less pain; saves money for himself and his employer; creates less inconvenience for himself, his family, his co-workers, and his employer; and is usually a good influence on all. Safety is contagious and is most easily caught by example.

Teaching of safety can be aided by dramatizing accidents. Posters listing the dire results from accidents, with accompanying pictures, are good. Analyses of the causes of accidents with a picture and legend make good posters. Include the cost to the victim, his family, his employer, and others involved; also the cost of replacing or repairing equipment.

Three-dimensional displays are very effective, from the wrecked car hanging at the sharp turn in the road, to a display of footwear to be worn in anesthetizing locations—conductive soled shoes, the correct way to wear conductive booties, strips and other appliances. Use red tape, for instance, to show the path of static electricity.

Bulletin boards can be used to advantage so long as they are kept up to date, changing the material frequently. This is true whether such a board serves just one department or the whole hospital.

Safety education can be dramatic in the classroom or in the regular safety committee meetings by dramatizing a particular accident taken from the hospital's incident files:—Writing a skit, including the victim's co-workers, family, insurance adjuster and others. This is a form of role playing, and is quite effective so long as the main and contributing causes of the accident are emphasized.

Sources of safety information and material are the American Hospital Association, 840 North Lake Shore Drive, Chicago 11, Illinois (limited supply) and the National Safety Council, 425 North Michigan Ave., Chicago 11, Illinois. (The inquiry should be directed to the Hospital Safety Service.)

Miss Aberg is A.A.N.A.'s representative on the N.F.P.A. Committee on Hospital Operating Rooms.

Any questions pertaining to hospital safety may be directed to the Executive Office. Answers will be included in this section in future issues.

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Abstracts

Roszkowski, A. P.: A pharmacological comparison of therapeutically useful centrally acting skeletal muscle relaxants. J. Pharmacol. & Exper. Therap. 129: 75-81 (May) 1960.

"Since the early studies of Berger and Bradley . . . describing the skeletal muscle relaxing properties of mephenesin . . . presumably through its action on the central nervous system, a large number of chemical agents have been prepared with the intention of simulating the pharmacological action of this agent. . . . This agent has several shortcomings, among which are its short duration of action and its obvious lack of potency. Subsequent to the discovery of mephenesin a number of drugs have been described pharmacologically which in some respects are improvements over this drug. . . .

"One of the purposes of our study was to compare all these agents [mephenesin carbamate . . . meprobamate . . . methocarbamol and zoxazolamine]. . . . A second purpose of this investigation was to describe the properties of another therapeutically useful benzazole, chlorzoxazone. . . .

"Zoxazolamine and chlorzoxazone appeared to be the most potent agents in inducing paralysis especially by the parenteral route. Meprobamate was a potent paralytic drug when administered orally. All the drugs tested with the exception of mephenesin were relatively long-acting compounds.

"Anticonvulsant studies carried out with these drugs indicate that chlorzoxazone possesses potent anti-strychnine activity while having virtually no protective action against pentylenetetrazol. Meprobamate displays potent anti-pentylenetetrazol activity and has relatively weak protective potential against strychnine. This suggests that chlorzoxazone may act principally at spinal levels and meprobamate at supraspinal levels."

Hyman, Chester and Winsor, Travis: Physiological basis for the clinically observed circulatory effects of isoxsuprine. Acta Pharmacol. et Toxicol. 17: 59-68, 1960.

"Indiscriminate use of 'general dilator' agents in the treatment of peripheral vascular disorders is contraindicated because of the possible diversion of blood flow away from already ischaemic tissues. . . . Several workers have recently reported successful treatment of night cramps, intermittent claudication and various vaso-spastic states with isoxsuprine. . . .

"Thirty patients with peripheral arteriosclerosis involving the lower extremities and 10 normal subjects were studied. The patients had mild to moderate intermittent claudication and were all ambulatory. . . .

"The cardiovascular effects of isoxsuprine have been measured in man. There is an increase in muscle circulation of the calf, with a smaller increase in the skin circulation of fingers and toes. Cardiac output is increased slightly. The agent is well tolerated in doses of 10 mg. intravenously or intramuscularly, or in oral doses of 10 mg. three times daily, providing other hypotensive agents are not given concomitantly and providing a tendency toward postural hypotension from other causes does not exist already."

Keats, A. S. and Telford, Jane: Studies of analgesic drugs. V. The comparative subjective effects of oxymorphone and morphine. Clin. Pharmacol. & Therap. 1: 703-707 (Nov.-Dec.) 1960.

"Oxymorphone (14-hydroxydihydromorphinone), a recently produced morphine derivative, has proved to be a potent analgesic both in animals and man. Although it possesses high addiction liability and is capable of producing hypotension and respiratory depression, oxymorphone has continued to arouse interest because of its reported low incidence of undesirable gastrointestinal actions. . . .

"Two groups of female patients who were awaiting elective surgical operation, most commonly gynecologic, were the subjects of this study. . . . Except for the effects drunk feeling, heavy feeling, itching, and dry mouth, the frequency of all subjective effects was higher after oxymorphone than after morphine. These differences were statistically significant only for the traits sleepiness, nervousness, dizziness, nausea and vomiting. . . .

"Only volunteered information was recorded. Sight difficulty included double vision, difficulty in focusing the eyes, and extreme dizziness. A heavy feeling usually referred to the extremities but at times to the head

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Volume 55
ANESTHESIA ABSTRACTS
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or 'all over.' A hot feeling included most but not all the patients who perspired; some patients who perspired profusely did not complain of feeling hot. . . .

"It was evident that oxymorphone did not possess lesser subjective side action liability than morphine and the claim of lesser gastrointestinal side actions after oxymorphone was not substantiated in this study."

Krantz, J. C., Jr., Ling, J. S. L. and Kozler, Virginia F.: Anesthesia LXI: The anesthetic properties of the azeotropic mixture of trifluoroethyl vinyl ether (Fluoromar) and 1, 1, 2-trifluoro-2, 2, 1-trichloroethane (Genetron 113). J. Pharmacol. & Exper. Therap. 130: 492-496 (Dec.) 1960.

"In an effort to reduce the flammability limits and also the expense of production, Fluoromar was mixed with 1, 1, 2-trifluoro-2, 2, 1-trichloroethane (Genetron 113) with which it forms an azeotropic mixture. . . . These studies describe the pharmacologic properties of this azeotropic mixture (AZT) and certain characteristics of Genetron 113. . . .

"Fluoromar and Genetron 113 form an azeotropic mixture which produced anesthesia in the rat, dog and monkey. AZT evokes an anesthetic syndrome similar to Fluoromar. These studies indicate that anesthesia with AZT does not produce deleterious effects upon the liver function or cardiac rhythm of the dog and, upon perfusion, the heart of the frog. Histologic studies revealed no pathologic findings in the lung, brain, kidney or bone marrow of the rat after 1/2-hour and 1-hour anesthesia with AZT over 7-day intervals. The multiple bursts of cerebral electrical activity evoked by anesthesia with Genetron 113 is not uncommon with certain fluorinated hydrocarbons or ethers. It appears that the depressant action of Fluoromar in AZT is sufficient to abolish the aberrant cerebral electrical activity.

"AZT, in its vaporized form at body temperature, retains the approximate composition of the azeotropic mixture in the liquid state. The two agents appear to exhibit synergistic anesthetic activity. The mixture affords the advantage of absence of explosive hazard and lower production cost.

"These experiments indicated that AZT exhibited desirable anesthetic properties in a variety of laboratory animals and in our opinion warranted trial by man. One of us (J. C. K., Jr.) administered AZT by the open-drop method to a patient requiring dilatation and curettage. She was premedicated with atropine sulfate and secobarbital sodium. The induction was smooth and rapid. Respiration and blood pressure remained essentially unaffected. Abdominal relaxation was good. The anesthesia was maintained for 30 minutes. Recovery was uneventful and occurred within 2 minutes after the discontinuance of the agent. There was no nausea or vomiting postoperatively. Constant observation of the hands and feet revealed no tremors as we had occasionally seen in animals."

Goldstein, Leonide and Aldunate, Jorge: Quantitative electroencephalographic studies on the effects of morphine and nalorphine on rabbit brain. J. Pharmacol. & Exper. Therap. 130: 204-211 (Oct.) 1960.

"We have re-examined the problem of morphine - nalorphine action and interaction using a recently available apparatus which permits quantitative analyses of some of the parameters of the EEG. . . . All experiments were performed on adult male albino rabbits. . . . It has been found that the curves relating log-dose/effect are not the same for the effects of these drugs. The blocking action of nalorphine on morphine revealed an apparent all-or-none threshold of action and a proportionally longer duration of action for small doses of nalorphine than for large doses."

Dybing, Fred: The mode of action of decamethonium on neuromuscular transmission in cat. Acta Pharmacol. et Toxicol. 16: 291-296, 1960.

"It is now generally accepted that decamethonium may block neuromuscular transmission either by prolonged depolarization of the motor end-plate or by competition with acetylcholine. . . . The purpose of the work described in this paper has been to investigate the action of decamethonium on neuromuscular transmission in the cat by a different technique from that used by Jewell & Zaimis. . . . The preparatory operations were performed on 8 cats under ether anaesthesia. . . .

"During continuous intravenous infusion of decamethonium it was found that this drug had a dual mode of action on both the tibialis anterior and the soleus muscles. Shortly after the infusion of decamethonium had been begun, the neuromuscular block had the characteristics of a depolarization block. When the infusion of decamethonium had continued for some time, the neuromuscular block changed to a more curare-like block. This was found both in the tibialis anterior and in the soleus, but the change in mode of action occurred earlier in the soleus than in the tibialis."

Adamkiewicz, V. W. and Adamkiewicz, L. M.: Glucose and the dextran "anaphylactoid" inflammation. Am. J. Physiol. 198: 51-53 (Jan.) 1960.

"The glucan dextran, a polymerized glucose, when injected into rats induces the "anaphylactoid" inflammation. We reported that insulin administration sensitizes to this inflammation. Conversely, the lack of insulin, such as it exists during alloxan diabetes, totally inhibits the inflammation. However, in alloxan diabetic rats, the true hypoinsulinism results in a secondary relative glucose overdosage of the animal. The role of such an overdosage in the inhibition of the inflammation has been investigated now. . . .

"Rats overdosed with glucose, and displaying a lasting glucosuria do not undergo the anaphylactoid inflammation when injected with the glucan dextran. . . . The mechanism of insulin sensitization to the dextran anaphylactoid inflammation could be explained partly as follows. Insulin diminishes in various ways the amount of "free" monomolecular glucose in the organism of the rat, leaving the transport mechanisms freer to carry the glucan dextran across cell barriers, in larger amounts, to the sites of the anaphylactoid inflammation. The over-all effect of insulin is therefore a potentiation of the inflammation."

Legislation

Emanuel Hayt, LL.B., Counsel A.A.N.A.

Nurse Anesthetist and Doctors Absolved of Negligence in Administration of Spinal Anesthesia to Heart Case

These are two actions of tort for malpractice. In one, a physician and two surgeons are defendants, and in the other the defendant is a nurse. Verdicts in favor of all of the defendants were directed.

The evidence reveals the following. The defendants are Dr. John E. Shaw, an anesthetist; Dr. Lewis S. Pilcher, the senior visiting surgeon at the Newton-Wellesley Hospital in 1954 and Dr. Francis H. Earthrowl who at that time was resident surgeon at that hospital. The defendant Anne McEnaney, a graduate nurse, was employed at the hospital as a "nurse anesthetist."

The plaintiff in April of 1943, when he was about eight years old, was admitted to the Newton-Wellesley Hospital. The provisional diagnosis on admission was "Question [of] acute rheumatic fever." The final diagnosis at the time of the plaintiff's discharge was "Acute rheumatic fever without rheumatic heart disease." Between 1943-1954, although the plaintiff returned to the hospital a few times for other ailments and check-ups, he lived an active, normal life. "In high school he starred on the football team and was on the wrestling, basketball and baseball teams."

During the evening of November 4, 1954, the plaintiff, then aged twenty and a student at a business college, complained to his mother that he had indigestion and a pain in his side. Early in the morning of November 5, following an examination by his family physician, he was admitted to the Newton-Wellesley Hospital. Upon admission, the defendant Earthrowl and another doctor were of opinion that the plaintiff was suffering from appendicitis and that an appendectomy that morning would be necessary. At that time the plaintiff's mother told Dr. Earthrowl of her son's history of rheumatic fever. Dr. Earthrowl, in accordance with hospital procedure, informed Dr. Pilcher of his diagnosis, and Dr. Pilcher instructed him to "schedule the case tentatively for an appendectomy." At some time prior to 8 a.m. Doctors Pilcher, Earthrowl and Shaw "reviewed the patient's history and physical findings in the hospital record."

Prior to anesthesia Dr. Shaw determined the plaintiff's blood pressure but did not record it. He testified that he determined the blood pressure after anesthesia and that it was the same. At 8:10 a.m. Dr. Shaw turned the patient over to the defendant McEnaney (the nurse), informing her that a satisfactory level of anesthesia had been obtained and that he was satisfied with the plaintiff's condition. Thereupon, Dr. Shaw and Dr. Pilcher, who also was present when the anesthetic was administered, left the operating room, the plaintiff thereafter being under the care of McEnaney.

Sometime between 8:10 and 8:20 (8:15 according to her testimony), McEnaney took the plaintiff's blood pressure, but "couldn't get any reading"; there was no blood pressure. She administered oxygen pressure to the plaintiff and informed Dr. Shaw of the absence of blood pressure. He directed her to give the plaintiff an injection of neosynephrine, which she did. Dr. Earthrowl administered an "intravenous infusion." He attempted to ascertain whether the heart was beating, "but didn't hear any heart beat." Upon Dr. Pilcher's arrival in the operating room, he and Dr. Earthrowl decided that the plaintiff's thoracic cavity should be opened for the purpose of massaging the heart. Such an operation was performed by Dr. Earthrowl and as a result the plaintiff's heart action and blood pressure were "restored to a normal level." The appendectomy was then performed.

The results to the plaintiff of the cardiac arrest are not in dispute. Because of it, his brain failed to receive the necessary supply of oxygen and serious damage resulted. The prognosis was that the plaintiff would "have to be cared for as a dependent person as long as he lives."

The plaintiff argues that the three defendant doctors were negligent in choosing a spinal anesthetic. There is no evidence that this choice was bad medical practice in and of itself, and the plaintiff does not so contend. Rather, the plaintiff bases his claim of negligence on the fact that at the time of the operation he was suffering from heart disease, and on certain medical testimony and excerpts from a medical treatise. But we are of opinion that the jury would not have been warranted in finding that the plaintiff had a heart disease at the time of the operation. Upon the plaintiff's discharge from the hospital after his attack of rheumatic fever, the final diagnosis, as above stated, was "Acute rheumatic fever without rheumatic heart disease."

The plaintiff contends that Doctors Pilcher, Earthrowl and Shaw were negligent in entrusting certain supervisory duties to the defendant McEnaney. More specifically, the plaintiff argues that the defendant doctors could have been found to be negligent in entrusting the plaintiff to the care of McEnaney during the thirty minute period after the spinal anesthetic had been administered, as that is the most dangerous time, that is, the time when a sharp drop in blood pressure could occur.

McEnaney was a nurse with twenty years' experience in the field of anesthesia. There is no testimony to show that the conduct of the defendant doctors, in entrusting the plaintiff to a nurse with McEnaney's experience, after anesthesia had been obtained, was a departure from the accepted procedure then obtaining in the Newton-Wellesley Hospital.

We are of the opinion that a finding of negligence on the part of McEnaney was not warranted. She testified that it would be normal practice "to take and record blood pressure readings at five minute intervals." When she was placed in charge of the plaintiff at 8:10 a.m. she was told by Dr. Shaw that the plaintiff's condition was satisfactory. She testified that she obtained a blood pressure reading at approximately 8:15 a.m. While this evidence could be disbelieved, such disbelief would not establish the contrary; nor was there any evidence to the contrary.

Dr. Shaw testified that when he turned the plaintiff over to the nurse at 8:10 "he was satisfied with the condition of the patient." While what is meant by a satisfactory condition is not further explained, it is reasonable to infer that at least it meant that the plaintiff's heart was functioning satisfactorily. Such medical evidence as there was on the subject showed that a cardiac arrest generally occurs very suddenly and without any warning. Even if Dr. Shaw's testimony as to the plaintiff's condition at 8:10 is disbelieved, there is a lack of affirmative evidence warranting the finding that the plaintiff's condition was not satisfactory at that time. If, as we have indicated above, Dr. Shaw could not be found negligent in entrusting the plaintiff to McEnaney's care, he could not be held responsible for her negligence, if any there was, after she took charge.

(Ramsland v. Shaw et al., 11 CCH Neg. Cases 2d 122-Mass.)

Parrillo

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The THIRTY-FOURTH QUALIFYING EXAMINATION for membership in the American Association of Nurse Anesthetists will be conducted on November 18, 1961. The deadline for accepting completed applications including the transcripts is October 9, Notice of eligibility will be mailed about October 16.

Applications should be forwarded early enough to allow time to request transcripts and have them returned to the Executive Office before the deadline date.

Book Reviews

General Anaesthesia for Dentistry. By James McNaught Inglis, M.B., Ch.B., F.F.A.R.C.S. (Eng.), D.A., Consultant Anaesthetist, United Birmingham Hospitals; Lecturer in Anaesthetics School of Dental Surgery, University of Birmingham and Victor Campkin, M.B., B.S., F.F.A.R.C.S. (Eng.), D.A., Department of Anaesthetics, Queen Elizabeth Hospital, Birmingham. The Williams & Wilkins Co., Baltimore, Md., exclusive U. S. agents. Cloth. 88 pages, 1960. $3.00.

This concise text was written primarily for the dental undergraduate in England. However, this material will be of value to anyone concerned with general anesthesia for dental surgery.

A chapter on "The Balanced Technique" is discussed at length. The importance of avoiding anoxia is emphasized throughout the book. Indexed.

Cardio-Vascular Surgery. A Manual for Nurses. Edited by George H. Peddie, M.D., Surgical Staff and Frances E. Brush, R.N., Director of Nursing, The Methodist Hospital, Houston, Texas. G. P. Putnam's Sons, New York. Paper. 170 pages. 1961. $2.75.

This manual has been prepared as a guide for nurses caring for patients undergoing cardio-vascular surgery. Many excellent illustrations and drawings are used.

Of special interest to anesthetists will be the chapter on anesthesia in cardio-vascular surgery and the appendix, which includes a discussion of the pump oxygenator.

This manual is highly recommended to the nurse concerned with the cardio-vascular patient. A bibliography follows the text. Indexed.

Anesthesia and the Law. By Carl Erwin Wasmuth, M.D., LL.B., Staff Anesthesiologist, Department of Anesthesiology, Cleveland Clinic Foundation and The Frank E. Bunts Educational Institute, Cleveland, Ohio; Assistant Professor in Legal Medicine, Director of the Department of Medical-Legal Affairs, Cleveland-Marshall Law School; Member of the Ohio Bar, Cleveland, Ohio. Charles C Thomas, Springfield, Illinois. Cloth. 105 pages, illustrated, 1961. $5.00.

This monograph is another in the American Lecture Series. Many of the chapters were taken from original articles by the author, some of which were published in medical journals. Dr. Wasmuth is an anesthesiologist and also has a degree of Bachelor of Laws.

Among the subjects discussed are: Consent, Negligence, Agency, Liability of the Anesthesiologist and the Surgeon, Endotracheal Anesthesia, Spinal Analgesia, Cardiac Arrest, Hospital Tort Liability, Physician-Patient Privilege, and Medical Records and the Hearsay Rule.

Actual cases are included to illustrate certain aspects of the law as it pertains to the practice of anesthesia. This text will be of interest to anesthetists, especially the portions on master-servant relationship and liability, as it pertains to the nurse anesthetist. References follow each chapter. Indexed.

Classified Advertisements

NURSE ANESTHETIST—C.R.N.A. to work with Anesthesiologist in 100 bed hospital. College town. Liberal employment policies. Salary open. Contact L. E. Wells, M.D., Southside Community Hospital, Farmville, Va.

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REGISTERED NURSE ANESTHETISTS: 690 bed hospital, primarily surgical. Integral part of developing 236 acre Detroit Medical Center. Emergency surgery only on Saturdays. Salary commensurate with qualifications. Excellent personnel policies. Write or call Personnel Director, Harper Hospital, Detroit 1, Michigan.

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WANTED — Lady Nurse Anesthetist — Group of 7 Physicians and 7 Nurses — Salary open — Contact Albuquerque Anesthesia Service, Medical Arts Square, N. E., Albuquerque, N. Mex.

NURSE ANESTHETIST: Immediate opening to increase staff from 3 Anesthetists to 4. 90 bed hospital. Salary commensurate with experience. Write: Allie Mae Fruge, CRNA, Chief Anesthetist, Galveston County Memorial Hospital, P. O. Box 127, La Marque, Texas.

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NURSE ANESTHETISTS — For modern 800 bed Teaching Hospital with modern Operating Room facilities; liberal Personnel Policies; group life, hospitalization, retirement benefits. Pleasant working conditions. Salary commensurate with training and experience. Residence accommodations available. Write to Personnel Director, Grady Memorial Hospital, 80 Butler St., S. E., Atlanta 3, Ga.

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Francis

(Continued from page 134)

visit and continues at least through the recovery room period. The complications of anesthesia in children most commonly result from poor control of gas exchange and secondary circulatory distress. Whereas adults react more slowly to the stress of hypoxia, the child may die unless immediate corrective measures are taken. Airway difficulty can be prevented by utilizing endotracheal management in anticipation of difficulty. It is important to choose equipment and techniques suited to the size and vitality of the child.

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Berger, Olive L.: The Use of Respirators in the Immediate Postoperative Period. J. Am. A. Nurse Anesthetists. *27*:182, Aug. 1959.

Adriani, John: The Chemistry of Anesthesia. Springfield, Ill. Charles C Thomas, 1952.

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